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A TEXT-BOOK

OF THE

# PRACTICE OF MEDICINE

BY

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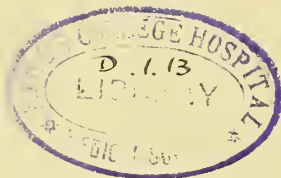
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## DISEASES OF THE CORONARY ARTERIES.

It has previously been noted that in pyemia and allied disorders septic emboli may block the branches of the coronary arteries, causing suppurative infarcts (acute circumscribed myocarditis).

It has also been shown that one of the chief effects of sclerosis affecting the coronary arteries is the production of *chronic myocarditis*. Sudden blocking of one coronary artery by an embolus causes instant death. In numerous instances in which death has occurred suddenly either thrombotic or embolic obstruction has been the only discoverable *post-mortem* lesion. In others the pathologic evidences of local or general atheroma have coexisted. Ligation or plugging of the coronary vessels in the lower animals causes arrhythmia or even an abrupt arrest of cardiac action; a partial or even slight reduction in the lumen of the coronary vessels by diminishing the supply of blood to the heart-muscle induces degenerations in the latter. Kronecker found that occlusion of the coronary arteries by injecting paraffin caused the heart to become irregular, even when it solidified in only the smaller branches, and stopped almost at once. The anatomic peculiarity of the coronary arteries in that they are end-arteries is to be noted, since it affords a ready interpretation of the usual effects following total or partial occlusion. According to F. H. Pratt, however, the vessels of Thebesius, which extend from the auricles and ventricles to the myocardial capillaries and coronary veins, may rarely maintain the nutrition of the heart-muscles even after occlusion of the coronary arteries.

The blocking of the terminal branches by emboli or by the more gradual formation of thrombi usually produces the so-called *anemic necrosis* or *white infarct*—a condition that richly deserves brief description :

**Anemic necrosis** (*anemic infarct*) is met with most frequently in the left ventricle and septum, which receive their blood from the anterior coronary artery. The involved areas are small and circumscribed, and present irregular margins that project slightly above the surface. Rarely the infarct is wedge-shaped. Its color is grayish-white or grayish-red, while the central portion is often distinctly white and firm; less frequently it breaks down into a soft detrital mass (*myomalacia cordis*). When softening does not occur the fibers in the affected area lose their nuclei, becoming first hyaline and subsequently sclerotic. The chief histologic changes are of two sorts: (*a*) the striæ of the muscle-fibers are lost, the latter becoming granular and breaking down; and (*b*) the fibers assume a homogeneous hyaline appearance, the nuclei having disappeared.

The *symptomatic* consequences of the lesions are often obscure and unreliable. Sudden death may take place, and rarely this accident may be due to rupture of the heart. Weak and irregular action of the heart, evidences of embarrassed circulation (especially in the cardio-pulmonary circuit, as shown by cough and dyspnea), and finally angina pectoris, are among the principal features observed. Death may ensue in the first attack. The paroxysms are presumed to be due to sudden occlusion of a branch of the coronary artery; but it should be

stated that occasionally in fatal instances of true angina pectoris a total absence of lesions, including emboli, has been noted. I desire to lay stress upon the medico-legal importance of coronary disease; it may be the only lesion found in cases of quick death.

## DEGENERATIONS OF THE HEART.

(a) **Fatty.**—The term “fatty heart” includes two pathologically distinct affections: (1) Fatty degeneration, in which the cardiac muscle-fibers have been converted into fat; and (2) Fatty overgrowth, in which an abnormal quantity of fat is deposited in and about the heart.

### FATTY DEGENERATION.

**Pathology.**—The condition may be either general or localized. Its most frequent seat is in the left ventricle, the papillary muscles and trabecule, first appearing as yellowish spots or stripes beneath the endocardium. The affected portions are light yellow or yellowish-brown (faded leaf) in color, due to an associated brown atrophy; they are also soft and friable, and are easily lacerated. The heart is enlarged, and often decidedly so if the process be general, and its walls lack firmness. The microscope reveals characteristic changes: the striæ and nuclei begin to fade, oil-drops and granules appear in the fibers, and finally the latter are occupied throughout by minute globules.

**Etiology.**—Fatty degeneration has already been mentioned as occurring in both the *primary* and *secondary* forms of *cardiac hypertrophy*. It is found also in association with fatty change in other organs in severe forms of *primary* and *secondary anemias*. It is most commonly encountered, however, in the *cachectic states* produced by such chronic diseases as carcinoma and phthisis, and in the course of *acute infectious diseases* of intense type, all of which may produce the condition. In poisoning by arsenic and phosphorus and in pernicious anemia it advances to a high grade. The various lesions of the coronary arteries previously considered bear a marked causal relation.

**Predisposing causes** are—(a) *age*—it being most common after forty years of age; (b) *sex*—it occurs somewhat more frequently in men than in women, notwithstanding the fact that there are predisposing influences at work in the latter that do not obtain in the male sex, such as childbirth and amenorrhea; and, lastly, (c) whatever may be its apparent etiology, it is invariably preceded by a defective nutritive supply to the muscle-cells; this may be dependent upon a narrowing of the lumen of the coronary vessels, or upon impairment of the oxygen-carrying power of the blood, as in the anemias. An excessive supply of glucose, glycogen, and nuclein may be a factor.

**Symptoms.**—The disease may exist in an advanced form without noticeable symptoms, though the conditions under which it is most liable to occur afford secure ground for suspicion. The evidences of *cardiac*

*enfeblement* are usually present, but in pernicious anemia the pulse may even be full and regular.

*Dilatation* is apt to supervene early, owing to the weakened state of the heart; and hence it is probable that many of the symptoms that have been ascribed to the fatty changes are in reality due to secondary dilatation. Among these are *palpitation*, *dyspnea*, a *small, irregular*, and somewhat *quicken*ed pulse, and *cool* and *clammy extremities*. The heart-sounds are weak, as a rule, and the action of the heart often irregular; later the physical signs of dilatation are almost invariably present. *Dropsy*, however, is rare in uncomplicated cases. Sometimes sudden, great physical exertion produces equally sudden dilatation, whereupon a canter rhythm and an apical systolic murmur speedily develop. In most instances, however, the symptoms are more gradually brought to light. *Breathlessness* on exertion is often a striking feature, and syncopal attacks are sometimes troublesome. The *pulse*, in consequence of irritation of the inhibitory center in the medulla, often becomes greatly retarded, dropping from the normal rate to 30 or 40 beats per minute, and, in rare cases, to 10 or 12 beats. The fatty *arcus senilis* is devoid of diagnostic value. There are frequent attacks of *cardiac asthma* in the mornings, and these are apt to be accompanied at intervals by *angina pectoris*. *Disturbance of the intellect*, sometimes taking the form of maniacal delusions, may come on and persist. Syncopal attacks occur. *Pseudo-apoplectic attacks*, such as have been described (*vide* Chronic Myocarditis), are also concomitants that point to disturbance of the cerebral circulation. *Cheyne-Stokes breathing* is among the later manifestations, and I have noticed that these symptoms often occur together. Epileptiform attacks resembling *petit mal* may arise.

The **diagnosis** is sadly obscure. The history, the age of the patient, and the symptoms of cardiac weakness and subsequent dilatation, together with retardation of the pulse, apoplectic attacks, and Cheyne-Stokes breathing, in the absence of precedent hypertrophy merely justify a probable diagnosis. With a clear history and the presence of the more significant symptoms, including the signs of dilatation following hypertrophy, fatty changes may be inferred with some degree of assurance, although a positive statement of opinion should be withheld.

The **prognosis** is as varied as the etiology. Death may come quickly, though oftener the end is reached in a gradual manner, the signs and symptoms of advanced dilatation dominating the closing scene.

**Treatment.**—The cause in each individual case should be determined with as much precision as possible, and when ascertained a bold attempt should be made to remove it. This course often places the patient in the most favorable position for the successful treatment of the cardiac condition; and the method embraces many hygienic and dietetic considerations that assist in improving the nutrition of the cardiac tissue—one of the cardinal aims of a proper system of treatment. Anemia in one form or other plays an important rôle in the majority of the cases, and the particular variety present in each instance must determine the character of the remedies to be employed. In that large cate-



gory of cases occurring in certain cachexias (cancerous, tuberculous) the following formula has given gratifying results:

R. Acidi arsenosi,	gr. j (0.0648);
Ferri sulph. exsic.,	gr. xxx (2.0);
Strychninæ sulph.,	gr. j (0.0648);
Quininæ sulph.,	ʒj (4.0);
Papoid,	gr. xxx (2.0).

M. et ft. capsulæ No. xxx.

Sig. One after meal-time, t. i. d.

A frequent, irregular pulse and other signs of cardiac failure indicate commencing dilatation, and under these circumstances digitalis should be employed in small doses. When used with perseverance it is of the greatest service, and in the form of the powder or the aqueous extract it may be conveniently combined with the above prescription.

I believe that gentle indulgence in physical exercise and light gymnastics is beneficial, since it tends to invigorate the heart-muscle; it is to be increased in proportion to the manifest improvement in the patient's condition. It sometimes happens, however, that even gentle exercise is badly borne, and it should then be discontinued. Kinesitherapy, particularly the milder Swedish method of gymnastic exercises (alternating movements of resistance), increases the contractile power of the heart and at the same time lessens the peripheral resistance, and should be accorded a careful trial. I have been in the habit of advising daily inhalations of oxygen gas in this class of cases with good results. Recourse to massage is also in the line of sound practice, but the sittings should not exceed half an hour in duration at the start. The more *prominent symptoms* may require special measures. The syncopal and anginal attacks are to be handled in the manner indicated for the same symptoms in chronic myocarditis. For the pseudo-apoplectic attacks rest in the recumbent posture, with the head slightly elevated, is useful. Therapeutic agents, as digitalis, ammonia, and ether, may be used hypodermically to stimulate the heart; it is also good practice to withdraw from 12 to 24 ounces (355.0–710.0) of blood directly from a vein. If the arteries be hard and tense, nitroglycerin is of distinct service.

A strictly horizontal posture and the application of ice to the precordial region often quickly terminate the attacks of cardiac asthma, and spartein sulphate, with nitroglycerin, is worthy of a trial. Hot toddy and other diffusible stimulants are valuable adjuvants. Should these remedies fail, hypodermic treatment by morphin is to be adopted.

#### FATTY OVERGROWTH.

**Pathology.**—The characteristic change consists in a marked increase in the normal fat, particularly in the auriculo-ventricular furrows. This over-production of fat takes place to a greater or lesser extent in every obese person, and may become so excessive as to form a complete enveloping mantle measuring an inch or more in thickness. In these extreme grades the muscular fibers of the organ may, from too great pressure, undergo atrophy and thus become weakened. Dilatation

often supervenes, and the principal symptoms date from the time of its occurrence. In the cachexias of carcinoma and phthisis, and the general atrophy of old age, fatty overgrowth and fatty degeneration coexist.

The **diagnosis** rests upon the combined presence of marked obesity and cardiac enfeeblement. (For the **etiology** and **differential diagnosis**, see p. 654.)

**Treatment.**—I wish to advocate warmly the system of treatment introduced by Oertel, as I have seen excellent results from its employment. It should not be resorted to in chronic valvular disease, in the stage of broken compensation, nor in marked atheroma.

Oertel's method comprises three parts: (1) The reduction of the amount of liquid taken with the meals and during the intervals, the total for each day being 36 ounces (1064.0). Frequent bathing (including the Turkish bath in suitable instances) and pilocarpin are employed to promote free diaphoresis.

(2) The diet is composed largely of proteids, as follows: *Morning.*—A cup of coffee or tea, with a little milk—about 6 ounces (178.0) altogether; bread, 3 ounces (93.0).

*Noon.*—Three to 4 ounces (90.0–120.0) of soup; 7 to 8 ounces (218.0–248.0) of roast beef, veal, game, or poultry, salad or a light vegetable, a little fish; 1 ounce (32.0) of bread or farinaceous pudding; 3 to 6 ounces (93.0–186.0) of fruit for dessert. No liquids at this meal, as a rule, but in hot weather 6 ounces (178.0) of light wine may be taken.

*Afternoon.*—Six ounces (178.0) of coffee or tea, with as much water. An ounce of bread as an indulgence.

*Evening.*—One or two soft-boiled eggs, 1 ounce (32.0) of bread, perhaps a small slice of cheese, salad, and fruit; 6 to 8 ounces (178.0–236.0) of wine, with 4 or 5 ounces (120.0–148.0) of water (Yeo).

(3) Graduated exercise up inclines of various grades. The distance to be undertaken each day is to be carefully specified and frequently, though gradually, increased. A like plan is to be pursued with reference to the degree of inclination. This is the most important part of the system, since it directly invigorates the heart-muscles.

(b) **Brown Atrophy.**—A form of degeneration in which accumulations of yellowish-brown pigment-granules occur in the muscular fibers. The color exhibited by the heart-muscle is a reddish-brown, and in pronounced cases a dark-red brown. Brown atrophy is most commonly seen in the hearts of the aged, though also quite often in cases of chronic valvular disease that have reached an advanced period before the time of the fatal issue.

(c) **Calcareous Degeneration (Calcification).**—Calcareous infiltration of the muscular fibers of the myocardium has been noted, though very rarely. Somewhat more common are the bony callosities that result from the inspissation and calcification of the purulent contents of former myocardial abscesses (*vide* Circumscribed Myocarditis).

(d) **Amyloid Degeneration.**—This form of degeneration is rarely met with. It is limited to the blood-vessels and interstitial connective tissue, the muscular fibers escaping, and its causes are the same as those of amyloid degeneration of other viscera.

(e) **Hyaline Degeneration.**—This is sometimes seen in association with amyloid change. It also occurs independently in prolonged fevers (*hyaline transformation of Zenker*). The fibers are swollen, translucent, and homogeneous, and their striæ almost entirely disappear.

## CARDIAC ANEURYSM.

(*Aneurysm of the Heart.*)

A CARDIAC aneurysm may, in the first place, involve the whole diameter of the myocardium (aneurysm of the walls).<sup>1</sup> Secondly, it may merely implicate the valves, together with a few myocardial fibers (valvular aneurysm).

**Aneurysm of the Walls.**—This is not of frequent occurrence. Its most common seat, however, is the wall of the left ventricle near the apex; it is quite generally a sequel to chronic myocarditis, which, as before stated, occurs oftenest at this point. Anything that produces a decided localized weakness of the ventricular parietes (other forms of degeneration and endocardial and pericardial inflammations) may, however, lead to its development. In size cardiac aneurysms are exceedingly variable, and may either be very small, or as large as the average-sized head of an adult. As to form, two types should be recognized: (a) an equable dilatation of a part of the ventricular wall, and (b) the sacculated form, which communicates with the chamber by a comparatively small orifice. Layers of fibrin are often found in these aneurysmal dilatations as an indication of Nature's attempt at a cure, and occasionally they may completely efface the sac when the attempt is successful. In most aneurysms non-laminated blood-clots are also found. It must not be forgotten that, once an aneurysmal distention has begun, a straining effort may cause a sudden great increase of the dimensions or even rupture it. The structures adjacent to the gradually formed aneurysm exhibit fibroid overgrowth and other kinds of degeneration, these changes being secondary and most probably conservative processes.

**Diagnosis.**—Aneurysm of the myocardium has no characteristic features. Usually the symptoms and local signs of chronic myocarditis or dilatation are more or less conspicuous, but the presence of the aneurysm is not even suspected unless certain physical signs develop in the course of the former complaints. These are—a pulsating prominence in the apex-region that may even perforate the chest-wall, and a coextensive dulness. The abnormal area of dulness is best appreciated early by stethoscopic percussion, but unless peculiarly circumscribed the condition cannot be distinguished from hypertrophy or dilatation. The course of these cases is unfavorable, death ensuing (rarely) from rupture of the sac or (more frequently) from gradual cardiac exhaustion.

**Valvular aneurysms** sometimes arise in acute ulcerative endocarditis, which destroys the segmented endocardium and permits of dilatation as the result of the intracardial blood-pressure. They occur with much

<sup>1</sup> Of 87 cases collected by Pelvet, 57 were in this situation, and of 90 collected by Legg, 59.



greater frequency on the aortic than on the mitral valves. They are spheroid in shape, and project into the left ventricle when found at the aortic segments, and into the left auricle when at the mitral. Though usually single, they are multiple in a few instances. Rupture of these aneurysms is common, with the subsequent development of extensive valvular incompetency. They cannot be *diagnosed* during life.

## RUPTURE OF THE HEART.

THIS rare and serious accident may either be *complete* or *partial*. The term partial rupture implies laceration of the trabeculæ ventriculi, whereby the chordæ tendineæ are liberated, or, more seldom, of the papillary muscle. The muscular structure may be involved to a slight extent. Valvular incompetency is the consequence of partial rupture. Complete rupture consists in a solution of continuity of the total diameter of the myocardium.

**Pathology.**—The chief seat of rupture is the anterior wall of the left ventricle, though it may also occur in the right ventricle and in the auricles, but with great rarity. The rent runs parallel with the muscular fibers, and is to a certain extent the result of laceration, though chiefly of a separation, of the fibers. The fissural communication presents irregular edges, and at autopsy is seen to contain blood-clots; the pericardial sac is also occupied by coagula, often in great numbers. If pericardial adhesions have previously obliterated the cavity, the escaped blood-clots may occupy the pleural cavity. Histologic examination of the muscle-structure surrounding the fissure shows the characteristic changes of fatty and other forms of degeneration.

**Etiology.**—Both predisposing and exciting causes may be at work. The former are the more important and always obtain, and, named in the order of their frequency of occurrence, the predisposing factors are disease of the coronary arteries (thrombotic and infectious embolic processes which produce anemic necrosis and abscesses), fatty degeneration,<sup>1</sup> chronic myocarditis, parietal tumors, and parasites in the heart-wall.

The influence of *age* as a predisposing factor has not been determined; rupture of the heart usually occurs after the sixtieth year has been passed, however, for the reason that the myocardiac changes that cause rupture belong to that period of life. Males suffer somewhat more frequently than females. The exciting cause is, as a rule, some form of muscular exertion, though it may occur spontaneously during sleep.

**Symptoms.**—In the majority of instances rupture of the heart results in *sudden death*. Sometimes, however, the patient survives the accident for several hours or even for as many days. The symptoms are those of *internal bleeding*, in addition to *pain* that may be agonizing and is referred to the heart. The body-temperature falls, the skin surface becomes pale and cool, and it may be covered with cold perspiration, while the *pulse* grows small, very frequent, and finally almost vanishes. Occasionally gastro-intestinal symptoms and syncope tending to convul-

<sup>1</sup> According to Quain's statistics, about 75 per cent. of the cases are due to this cause.

sions appear in consequence of the irritation of the vagus centers due to cerebral anemia. The *physical signs* of cardiac failure rapidly develop, and, if the leak be not too large, those of pericardial effusion more gradually.

**Diagnosis.**—A certain diagnosis is rarely possible. Heart-anguish, rapidly progressive cardiac failure, the evidences of internal hemorrhage, and the speedy development of the signs of pericardial effusion should, however, always excite a strong suspicion of rupture, and in many cases suffice for a correct inference.

The **prognosis** is hopeless. When immediately fatal, death is the direct result of heart-shock; when delayed, the sad issue takes place in consequence of anemia of the brain or of compression of the heart by the blood that pours into the pericardial cavity.

**Treatment.**—*Prophylaxis* is of the utmost importance. In all conditions of the cardiac parietes in which this accident is liable to occur the physician should not fail to give ample warning of the dangers connected with muscular strain of whatever sort. If rupture has occurred or is suspected to have taken place, the patient must be put at complete rest in the horizontal position. Full doses of morphin should be given hypodermically, and the ice-bag locally applied. Warmth to the extremities may be useful, but applied to the heart-region can be only harmful. The use of pure cardiac stimulants will be attended with increased bleeding from the rent, but agents that relax the peripheral arterioles, such as nitroglycerin, may be employed with a view to diminishing the heart's labor without diminishing its power. Should the rupture be partial and the hemorrhage slight, the patient's life may be prolonged, or even saved, by keeping him at absolute rest for a long period or until Nature effects recovery.

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## MINOR AFFECTIONS OF THE HEART.

(a) **New Growths.**—Primary carcinoma or sarcoma is rare indeed. Metastatic growths occur, but are very rarely sufficiently large (except perhaps the colloid variety) to be detected by physical examination, or to give rise to symptoms. Very large tumors may weaken the heart-muscle, but this must be an extremely rare occurrence. Kayserlink reports a case of lympho-sarcoma with well-marked symptoms. The separation of portions of the tumor may, if of considerable size, block one of the valvular orifices and cause sudden death, or more minute portions, becoming released, may give rise to embolism in distant parts. Tuberculosis and syphilis have been considered elsewhere.

(b) **Parasites.**—Four forms may invade the heart-muscle—the tænia echinococcus, actinomyces, cysticercus cellulosæ, and the pentastomum denticulatum. The former two are alone productive of mischievous results. The echinococcus growths may attain to considerable dimensions and are often multiple; they are secondary to echinococcus-cysts in other organs. Their effects are produced in a purely mechanical manner unless fragments become detached, when they may excite embolic

lesions at different points in remote organs. Embolic abscesses have occasionally been observed, appearing like degenerations due to the *actinomyces fungus*.

(c) **Misplacement** (*Transposition of the Heart*).—During intra-uterine life the heart (and rarely all the other thoracic and abdominal viscera) may either be transposed to the right side of the thorax, or the fetal position—in the median line—may be retained. The sternum may be missing in whole or in part, and the heart, which now lies immediately beneath the skin, can be seen and felt as a throbbing tumor. Recently a man of about forty years applied at the Medico-Chirurgical Hospital in whom the lower half of the sternum was absent; his heart occupied a position in the median line directly underneath the skin, where its strong pulsations could be felt. The patient stated that it had given him no inconvenience.

Very exceptionally other anomalous positions are acquired during ante-natal development, and the heart may become displaced upward in the chest-cavity even to the neck or downward into the abdominal cavity.

(d) **Floating Heart**.—The structures that serve to maintain the heart in its normal anatomic relations may become weakened or unduly lax, in consequence of which the organ may exhibit increased motility.

### III. NEUROSES OF THE HEART.

#### PALPITATION.

**Definition.**—A more or less rapid action of the heart that is perceptible to the patient, and usually accompanied by an increased force of the cardiac contractions or a disturbance of the rhythm, and often also by precordial distress, anxiety, and dyspnea.

**Etiology.**—Chronic valve-disease and other organic affections of the heart seldom produce palpitation, numerous conditions outside of the organ being more frequently related causatively. Among these are—(1) Mental excitement, depression or emotion; (2) Anemia (from the local irritant action of the altered blood-state); (3) The acute infectious diseases, in which the toxins in the blood irritate the cardiac accelerating nerves; (4) Dyspepsia, even in robust-appearing persons (as in the gouty) who willingly or unwillingly commit dietetic errors. Special articles of diet may excite over-action (*e. g.* strawberries, shell-fish), the palpitation thus arising from reflex irritation being dependent upon gastric catarrh. (5) The use, and more especially the abuse, of tea, coffee, alcohol, and tobacco. These agents are injurious largely through their effects upon the nerves. (6) The female sex manifests a greater disposition to the complaint than the male, especially about the period of puberty and the menopause. In the male it is most common at or after the middle period of life, a time when the effects of the work and worry of life show themselves. (7) Disturbances of the ovaries and other pelvic organs may induce palpitation reflexly.

**Symptomatology.**—Cardiac over-action may, though rarely, be



constant, but, as a rule, it displays a definitely *paroxysmal* character. The *onset* is sudden, and immediately preceding the attack there are often a blanching of the face and a slowing of the cardiac action, symptoms due to the momentary inhibitory effect of the nerve-affections that cause the "palpitation." The patient's *perception* of increased force and rapidity of the heart's action is the essential symptom. The patient may complain of *throbbing sensations* and *palpitation*, with a normally acting heart, the symptoms being wholly subjective in character. *Mental anxiety* is common, and dyspnea, the latter symptom assuming curious phases. In a recent case of my own the patient would attempt at intervals of three to five minutes a forcible, long-drawn inspiration, which would sometimes successfully relieve his respiratory difficulties for a while.

**Physical Signs.**—*Inspection* shows the impulse to be somewhat diffuse and forcible. Visible throbbing of the superficial vessels is also common. The *finger-tips* easily appreciate the increased strength of the impulse. At the wrist the pulse, though strong and full, as a rule is rapid, the rate varying from 120 to 160 per minute. *Percussion* may show the area of cardiac dullness to be enlarged, while *auscultation* reveals louder sounds than the normal. The attack is usually of brief duration—but a few minutes—though sometimes it may last for hours or days.

Attention should here be called to the *irritable heart* described by DaCosta—a form of palpitation common among young soldiers during the late Civil War. It was caused partly by mental excitement and partly by inordinate muscular exertion. A minor part in its production was also played by the diarrhea that was so often present. The leading symptoms were palpitation, a very frequent pulse, dyspnea, and cardiac pains of varying intensity.

**Differential Diagnosis.**—Nervous palpitation must be distinguished from the comparatively rare cases in which the heart contracts rapidly and irregularly, but does not excite subjective sensations. Some of the latter instances are to be looked upon as physiologic, while others are due to exhaustion and other causes. They do not constitute cases of palpitation, since they are unperceived by the patient, but are in reality cases of either tachycardia or arrhythmia.

Palpitation due to *chronic valve-disease* should also be differentiated from the purely nervous form. Here chief reliance is to be placed upon the presence of a murmur and other physical signs during the intervals between the attacks. *Anemic murmurs* are sometimes present, and must not be confounded with those of *organic nature*.

**Prognosis.**—The condition is free from real danger to life. Most authors, however, are agreed that cardiac hypertrophy may be a sequel.

**Treatment.**—The chief indications for treatment are—(1) *The arrest of the paroxysm.* The patient must be put at absolute rest in bed in a large, well-ventilated, darkened chamber, and his clothing loosened so that the respiration is unimpeded. Pressure upon the vagus in the neck or upon special points on the abdominal parietes (the ovarian region in particular) sometimes arrests the attack promptly. In my own hands the best results have been obtained from the application of the ice-bag to the precardial region. If this does not succeed in cutting short the paroxysm in the course of a couple of hours, the ice-bag should be removed every third hour. In conjunction with this measure the

patient should be told to take large draughts of cold water or to swallow bits of ice. On the other hand, I have observed a few instances which were speedily relieved by the ingestion of hot and somewhat stimulating drinks. It is, however, not possible to formulate general rules that will be applicable to all cases. Kinnear<sup>1</sup> treats cardiac palpitation by applying cold over the sympathetic ganglia of the spinal cord.

Among the many therapeutic measures that have been employed, morphin alone has given quite constantly good results, and particularly when administered hypodermically. However, before employing morphin, other sedatives and narcotics should be tried, such as the bromids (in large doses), hyoscyamus, hyoscin, and camphor monobromate. In neurasthenic and hysteric subjects the bromids and the preparations of valerian are highly serviceable. The tincture of valerian or the elixir of valerian ammoniate may be used, and I have found the following capsule of great utility:

R <sub>x</sub> . Zinci valerianat.,	gr. x (0.648);
Strychninæ sulph.,	gr. $\frac{1}{3}$ (0.0216);
Ext. sumbul.,	gr. x (0.648);
Ext. hyoscyami,	gr. v (0.324);
M. et ft. capsulæ No. x.	

Sig. One after meal-time.

If a special article of diet or an overloaded state of the stomach is the cause, an emetic may be given and the attack thus speedily controlled. Oxygen-inhalations have been warmly advocated.

(2) *To prevent a recurrence of the paroxysms*, the causal conditions, some of which may long antedate the occurrence of palpitation, must be removed, if this be possible. All exciting factors must also be avoided. The use of tea, coffee, and tobacco must be discontinued, and alcohol should be allowed only in small amounts. The general health must be considered, and anemia, chlorosis, neurasthenia, or hysteria must each receive appropriate treatment when present. When cardiac palpitation occurs in neurasthenia and hysteria the Weir Mitchell rest-cure should be advised, its results often being strikingly good if rigidly practised. Galvanism of the pneumogastric is sometimes useful, the positive pole being placed under the angle of the jaw, and the negative lower down, over each side of the neck. The removal of certain local conditions that sustain a causal relation, as gastric catarrh or intestinal parasitic diseases, is a question that must not be overlooked. If the heart be weak, digitalis may be exhibited for a long time in small dose. I have observed good effects from the use of baths (carbonated).

## TACHYCARDIA.

(*Tachycardia Paroxysmalis*; *Synchopæxia*; *Rapid Heart*.)

**Definition.**—A rapid movement of the heart occurring in paroxysms of variable duration, and directly dependent upon either paralysis of the pneumogastric or stimulation of the sympathetic nerves. It is

<sup>1</sup> *Med. Rec.*, July 16, 1898.

not dependent upon chronic valvular disease, nor upon other gross organic lesions, nor is it generally accompanied by notable subjective sensations. Martius believes that the condition is attributable to sudden dilatation.

**Pathology and Etiology.**—It occurs as a physiologic condition in a certain proportion of the human family; in such cases the pulse may range from 90 to 100 beats per minute or over. Certain individuals can increase the pulse-rate by their own volition. The pathologic forms are divisible into—(1) Essential or neurotic tachycardia, and (2) Symptomatic tachycardia.

(1) **Neurotic Tachycardia.**—The causes of this variety are identical with many of those that excite palpitation; hence these cardiac neuroses are often associated. Thus, among disposing factors are hysteria, anemia, neurasthenia, chlorosis, and toxic agencies (tea, coffee, tobacco, the poisons of fevers). Violent exercise, intense mental agitation, fright, grief, and other forms of shock are determining influences. Not a few cases are met at or about the menopause.

(2) **Symptomatic Tachycardia.**—The lesions that induce this form are—(a) *central* and (b) *peripheral*. In the former group are especially to be placed tumors, clots (due to hemorrhage), and softening of the medulla and cord; and in the latter, tumors, aneurysms, enlarged lymph-glands (which paralyze the vagus by exerting pressure upon it either in the neck or thorax), and neuritis, affecting the pneumogastric nerve. The latter lesion may be associated with polyneuritis (alcoholic or infectious). Rapid heart may be due to *reflex irritation* (gastric, intestinal, arterial, uterine, ovarian), or gastro-intestinal intoxication.

**Symptoms.**—The clinical picture in most instances of the complaint is made up of recurring paroxysms of heart hurry (*paroxysmal tachycardia*). These attacks come on with great suddenness, and, as a rule, without prodromes or forebodings. If the latter occur, they consist of vertigo, tinnitus, and a sense of impending danger, and sometimes persist to the end of the attack. With the onset of the paroxysms the *cardiac movements* leap to 150, 175, 200, and 250, or even

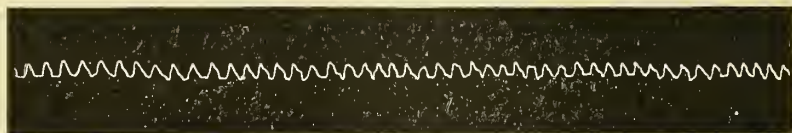


FIG. 55.—Radial pulse during an attack of paroxysmal tachycardia.

to 300 beats per minute. The *pulse* is feeble, small, readily compressible as a rule, and sometimes irregular (Fig. 55). Rarely it is full, strong, and of good tension. The *respiration* may or may not be increased in frequency, but dyspnea is not common. Respiratory oppression and smothering are seldom witnessed in genuine tachycardia. At first pale, the *skin* soon becomes flushed, and the *countenance* may wear an anxious expression; but unless “palpitation” is associated there are no symptoms present that denote an intense degree of suffering. In many cases the patient is not conscious of palpitation, or there may be a sense of slowing of the heart, when in reality the cardiac con-



tractions may be increased to 200 or more ; this is *typical tachycardia*. In a chlorotic girl I found that the pulse-rate increased to 200 beats, and lasted for a few minutes at each visit to my office. During the intervals between the visits the pulse was apparently normal in frequency. H. C. Wood reports a truly remarkable case occurring in a physician in his eighty-seventh year, who has had attacks at intervals since his thirty-seventh year. Following a sudden onset, the pulse rises quickly to 200 beats per minute. The attacks can be averted by the taking of ice-water or strong coffee.

**Physical Signs.**—A diffuse, rapid, and sometimes irregular impulse may be observed on *inspection* and *palpation*, but seldom is there an enlargement of the heart. The *sounds* are slightly modified, the first being accentuated and the second aortic greatly diminished in intensity, owing to the lessened amount of blood thrown into the aorta with each ventricular systole ; the intensity of the second pulmonic, however, may be increased. An apical *systolic murmur* is occasionally audible. The carotids pulsate, and on auscultating over them a murmur is sometimes heard. The *duration* of paroxysmal tachycardia varies from one to two or more decades.

**Diagnosis.**—I would restate the fact that a high pulse-rate (200 or over a minute) and an absence or only a slight sense of palpitation or rapid heart-action are the distinctive features of true tachycardia. In *palpitation* (previously considered) the pulse-rate is not usually so high as in tachycardia, while the associated phenomena of dyspnea, precordial constrictions, smothering, and painful anxiety are correspondingly more pronounced.

**Prognosis.**—In the majority of cases no serious impairment of the general health follows, though the course is exceedingly chronic and recoveries are comparatively rare. When symptomatic tachycardia is due to lesions that are removable, it is often curable, though not invariably so. In sufferers who are advanced in years, however, the cerebral vessels may rupture. Boveret analyzed a number of cases, 4 of which proved suddenly fatal in consequence of heart-failure. In forecasting the issue during the paroxysm these serious accidents must be considered.

The **treatment** is to be conducted on precisely the same lines as those advanced for "Palpitation" (*vide* p. 664).

## BRADYCARDIA.

(*Bradycardia*.)

**Definition.**—Slowness of the pulse. The condition may be physiologic, the rate of the pulse being sometimes 60 or less, and very rarely as low as 40 per minute during perfect health.

All cases of pathologic bradycardia fall naturally and conveniently into two groups : (1) those that are secondary to other complaints (*symptomatic bradycardia*) ; and (2) those that are due to, or associated with, a neurosis.

**Pathology and Etiology.**—**Symptomatic Bradycardia.**—(a) Arising during convalescence from acute infectious diseases, especially *pneu-*

*monia, typhoid, diphtheria, influenza, and acute rheumatism.* According to Riegel, who analyzed 1047 cases in which the pulse-rate was less than 60, the *acute fevers* must be awarded the first place among the causal factors. I have met with 3 cases of diphtheria in which the pulse in convalescence fell to 30 a minute. That such instances are, as Traube contends, due to exhaustion is true of some cases, but not of all, and doubtless there are other changes in a certain proportion. The slowing of the pulse that is observed after premature or full-time delivery is similarly produced. (b) The second place belongs easily to gastro-intestinal and hepatic disorders (*chronic gastro-intestinal catarrh, ulcer, or carcinoma of the stomach*). (c) Brachycardia occurs in diseases of the circulatory system—in *coronary disease*, fibroid and fatty myocardial change, most frequently; and chronic valvular disease much less frequently, if we except aortic stenosis. (d) Pulmonary complaints (emphysema and asthma). (e) Toxic agencies, as in jaundice, blood-poisoning, alcoholism, the unwonted use of tea, coffee, tobacco, and a few drugs (*e. g.* digitalis, strophanthus). (f) Constitutional affections (anemia, chlorosis, gout, diabetes). (g) Rarely skin-diseases and affections of the sexual organs, and commonly myxedema, are associated with brachycardia. In various organic nerve-affections (apoplexy, meningitis, epilepsy, tumors of the cerebrum, and the medulla in particular, injuries and diseases of the cervical portion of the cord). In such cases the brachycardia is due chiefly to direct or reflex irritation of the center or of the peripheral portion of the vagus system. Indeed, brachycardia is produced in one or other of these ways, except in those cases in which it is brought about by exhaustion of the automatic motor apparatus of the heart.

(2) **Brachycardia associated with a neurosis** may be found to be marked in *epilepsy*; less so in *hysteria, melancholia, mania*, and *general paresis of the insane*. It precedes palpitation.

**Symptoms.**—The sole characteristic symptom is the *slow action* of the heart, and this may either be temporary or permanent. If *paroxysmal*, both the onset and termination are apt to be sudden. A slow emergence is, however, more common than a slow beginning, though a small group of prodromes appears, comprising vertigo, tinnitus, and a sense of impending danger. During the paroxysm the patient may repeatedly suffer from *syncopal attacks* or become *unconscious* for hours at a time; *physical prostration* may also be marked, and especially when secondary to chronic valve-disease. The *pulse* is weak and small, and the beats per minute vary from 50, 40, 30, 20, to 10, or even 8. When the condition arises in the course of organic valve-lesions the *cardiac contractions*, as a general rule, may be increased in power, though greatly reduced in frequency. Thus, I observed this occurrence in a patient under my care at the Philadelphia Hospital suffering from a double mitral lesion and aortic constriction. The pulse fell from 70 to 28 per minute, but the systole was more powerful than before brachycardia was developed. The pulse at the wrist does not show the rate of cardiac contractions (when the heart is weak), since the latter do not always emit a pulse-wave that can be detected at the wrist; hence the heart-action must be noted by auscultation, and the rate compared with that of the peripheral pulse. The *impulse* and the *heart-sounds* are feeble.

**Diagnosis.**—A pulse below 48 beats per minute, with corresponding slowness of the systole, suffices for a certain diagnosis.

The **prognosis** is governed by the cause, being very grave in cerebral and advanced cardiac diseases. When fatal, sudden death is the rule.

**Treatment.**—Rest in the lying posture, particularly if the condition has come on in organic heart-disease, and such remedies as atropin, strychnin, caffein, nitroglycerin (in small doses), and ammonia are to be given a trial. If the ventricular contractions are very feeble and not below 30, small doses of digitalis will be found useful, though the effect must be closely watched. In the intervals between the attacks the general health must be improved and the causal states eradicated.

## ARRHYTHMIA.

(*Irregular Heart- and Pulse-beat.*)

(1) THE irregularity may affect only the *volume* and *force* of the pulse. Here the intervals between the beats are equal, but in regard to fulness and strength the beats are unequal. Instances of irregularity in the volume and strength of heart-beats may give rise to the condition known as *pulsus alternans* (Traube), in which fuller and stronger pulse-beats regularly alternate with those of lesser volume and strength (see Fig. 56). (2) **Irregularity in Time.**—(a) Intermittent heart-beat. This



FIG. 56.—Pulsus bigeminus alternans (Eichhorst).

is but an exaggerated degree of the first variety, and signifies a missed or dropped beat. This occurs at irregular intervals in most of the cases, though sometimes a cyclical irregularity is observed—*i. e.* every second, fourth, sixth, or eighth beat being lost. (b) Twin-pulse (*coupled beats, allorhythmia*). When two beats follow each other quickly (the diastole being shortened), and the next two not so quickly (the diastole being lengthened), we have produced the *pulsus bigeminus*. The first and second beats may be of equal strength, but often the second is relatively feeble. This is best determined by auscultation of the heart, since the second systolic contraction (of the ventricle) may indeed be so weak as not to give rise to a palpable beat at the wrist. I have frequently observed the *pulsus bigeminus* in mitral disease. With respect



to the diastole, the approximated pulsations may be in blocks of three (*pulsus trigeminus*), or even of four (*pulsus quadrigeminus*). (3) **Combined irregularity** of time and volume. Whilst the forms of irregularity described above should be distinguished from one another, this is not always practicable, particularly in the last stages of valvular affections and in the acute infectious diseases—conditions in which the heart-muscle fails in consequence of degenerative changes. (4) The **paradoxical pulse** of Kussmaul also consists in irregularity of volume, strength, and time, though not indicative of so great peril as the preceding. It is dependent upon the act of inspiration—"normal as well as forced"—the beats during inspiration being more rapid, though weaker, than during expiration. This is met with in chronic adhesive pericarditis, in cases of pressure upon the root of the aorta by bands, in pleuro-pericarditis, and in a very weak heart. (5) **Delirium cordis** is a term very appropriately given to great irregularity and inequality of the pulse-beats. It is seen in extreme dilatation and advanced exophthalmic goiter. (6) **Embocardia or Fetal Heart-rhythm**.—There is a shortening of the long pause with a striking similarity of the first and second sounds, as in the fetal heart. I have already pointed this out in connection with dilatation, though it also sometimes attends the advanced stages of grave fevers. (7) **Cantering Rhythm** (*bruit de galop*).—The sounds simulate the triple footfall of a horse at a canter. The interpolated sound is due to a reduplication of the second, though rarely it is the first that is doubled instead. It is developed in the hypertrophy of arterio-sclerosis and Bright's disease, in profound anemias, and in the myocarditis of certain acute infectious diseases. (8) **Tremor Cordis**.—By this is meant a ventricular systole so rapid as to be evidenced by mere vibrations.

**Etiology**.—Baumgarten's classification of the causes of arrhythmia (quoted by Osler) is the best, and is here given:

(1) Those due to central—cerebral—causes, either organic disease, as in hemorrhage or concussion, or more commonly psychological influences.

(2) Reflex influences, such as produce the cardiac irregularity in dyspepsia and diseases of the liver, lungs, and kidneys.

(3) Toxic influences. Tobacco, coffee, and tea are common causes of arrhythmia. Various drugs, as digitalis, belladonna, and aconite, may also induce it.

(4) Changes in the heart itself. (a) In the cardiac ganglia. Fatty, pigmentary, and sclerotic changes have been described in cases of this sort, and these may have an important influence in producing disturbances in the rhythm, but as yet we do not know their exact significance. They may be present in cases that have not presented arrhythmia. (b) Mural changes are common in conditions of this kind. Simple dilatation, fatty degeneration, and sclerosis are most commonly present, the two latter being usually associated with sclerosis of the coronary arteries.<sup>1</sup>

**Symptoms**.—Arrhythmia, particularly when functional or of reflex origin, may exist for years together, without associated symptoms referable to the heart, and hence is often discovered accidentally. When it is combined with palpitation or extreme weakness or dilatation of the organ, it is apt to arrest not only the attention of the observer in many instances, but also that of the patient.

<sup>1</sup> *Transactions of the Association of American Physicians*, vol. iiii.

**Physical Signs.**—In given cases the cause will be found to govern the character of the physical signs, which are often scanty or sometimes practically wanting. Those usually present have been indicated in speaking of the different varieties.

**Diagnosis.**—*Palpation* and *auscultation* of the heart while examining the pulse are matters that should never be neglected if reliable results are to be obtained. It is especially in this class of cases that the sphygmograph renders invaluable aid. Sphygmograms will often show the kind and degree of arrhythmia when all other means of examination have failed, and also distinguish marked diastolic irregularity from irregularity.

It is important to *differentiate* functional arrhythmia or that of reflex origin from arrhythmia due to more or less grave myocardial disease. Important information is supplied by carefully reviewing the varied etiological factors that produce the functional form, and by close observation of the cardiac symptoms.

The **prognosis** is variable. A gentleman with whom I am acquainted was rejected by a life-insurance company twenty years ago on account of occasional slight arrhythmia, though he is still in active business life and apparently in vigorous health. When the myocardium becomes involved, as occurs in chronic valvular or coronary disease or in the acute infectious diseases, the prospect is gloomy; on the other hand, when it is functional or due to other causes outside of the heart itself, the course pursued is as a rule favorable. When the second sound follows closely the first (marked abbreviation of the systolic pause) it is a serious indication.

**Treatment.**—There are many cases of the more benign form in which little, if anything, can be accomplished save to benefit the patient's general health, and this, I take it, is of paramount importance. Removal of the causal forces, as tea, coffee, alcohol, indigestible food-stuffs, conditions acting in a reflex manner, must be executed promptly. When the condition is due to changes in the heart-structure, cardinals in addition to the general tonics should be prescribed. I prefer strychnin, arsenic, and the dried sulphate of iron in combination. Nitroglycerin is of service if the arterial tension be high. If the arrhythmia be due to excessive cardiac dilatation, digitalis should be employed. In purely functional cases, in which there is a predominating neurotic element, the subjoined formula has been useful in my hands:

R. Ferri valerianatis,  
Zinci valerianatis,   *āā.* gr. xxx (1.94);  
Strych. sulph.,           gr. j   (0.0648);  
Pulv. digitalis,           gr. viij (0.518).

Ft. capsulæ No. xxx.

Sig. Take one after meal-time.

## ANGINA PECTORIS.

(*Stenocardia, Breast-pang.*)

**Definition.**—A paroxysm of violent precordial pain extending into the neck, back, and arms, and at times attended by a sense of impending

death. It scarcely deserves to be classified as a separate disease, being merely symptomatic of several cardiac lesions already described.

**Pathology.**—Concerning the pathogenesis of angina, we possess few, if any, positive data. Many theories have been advanced, but to adduce them here could serve no useful purpose, and conclusive *post-mortem* evidence in support of the various theories that have been and are advocated is, as yet, wanting. Neither does any single hypothesis fit the grouping of symptoms so constantly observed in cases of angina. It is to be recollected, however, that it is a neurosis affecting the cardiac sensory filaments that are given off chiefly from the pneumogastric, and in many cases the vasomotor apparatus is also involved. Nothnägel has described a form distinct from the above in which the vasomotor apparatus is principally involved (*angina pectoris vasomotoria*).

**Etiology.**—Cases of angina unassociated with *arterial sclerosis*, *hypertrophy*, *aortic regurgitation*, or adherent pericardium, are rarely encountered. It is quite probable that with few exceptions sclerosis of the aorta and coronaries is present, and predisposes to the disease under discussion. This view also receives some degree of color from the fact that angina usually occurs after the *fortieth year*, and principally in the *male sex*. It may be a sequel of influenza. The *determining factors* of the attack are undue exertion and mental emotion.

**Symptoms.**—The *paroxysm* begins quite suddenly during the action of one or other exciting cause. There is excruciating pain of a grip-like character, affecting the entire chest and rendering the body motionless. The pain *radiates* most frequently to the left shoulder, though also at times to the right, and thence to the back, neck, and down the arms to the fingers. *Coldness* and *numbness* of the fingers or in the precordial area may be present. Not less agonizing than the pain is the awful sense of *impending death*. The *countenance* is frequently pale, and may assume a leaden hue, and is usually bathed in cold perspiration. The *respirations* are exceedingly shallow or even temporarily arrested, and the patient's anxiety is extreme. The *heart's action* may be regular, and the arterial tension, as shown by the pulse, is generally increased. The *duration* of the paroxysm varies from a few seconds to a minute or two, and after the pain is over gaseous eructations, vomiting, or the discharge of a large amount of clear urine may occur. With the cessation of the attack comes instant relief from the cardiac symptoms. On making a careful examination of the heart subsequent to the seizure there may be an utter *absence of signs*, and, though there is weakness, this soon disappears. The attacks may *recur* at intervals varying from a few days to many years. I have usually found that cases associated with aortic regurgitation give the shortest intervening periods as a rule.

In *angina vasomotoria* the pain in the heart-region is preceded for a few minutes by pallor of the face, coldness and stiffness of the limbs, due to spasm of the peripheral vessels.

**Pseudo-angina.**—This is also a *paroxysmal* affection, that may be associated with hysteria, anemia, or neuropathic inheritance, and is occasionally produced by infectious diseases, poisons, and the like. Its symptomatology is given below.

**Diagnosis.**—The characteristic events are a sudden, most intense pain in the substernal and left parasternal regions with marked constrict-



tion of the chest, the peculiar manner of radiation of the pain, and the sense of impending death. Less diagnostic, though of considerable value, are the brevity of the attack, the sudden onset and cessation of the pain, the age and sex, and the anxious, moistened features. There are also lighter forms in which one or more of the diagnostic phenomena above described are absent. If they occur between the ages of forty and sixty years in persons in whom either arterial sclerosis or aortic regurgitation is present, this disease should be thought of; and after the exclusion of certain complaints in which paroxysmal pain is prominent, such as gastralgia and locomotor ataxia, the diagnosis of angina becomes reasonably certain.

The distinction between true and pseudo-angina pectoris is not always easily drawn, but the most important points for discrimination may be found in the tabulated statements below:

## ANGINA PECTORIS.

Etiology indeterminate, though generally associated with arterio-sclerosis (including coronary disease) or aortic regurgitation.

Occurs after the fortieth year, usually in males.

Paroxysms, provoked by undue exertion or mental emotion, are rarely periodic and nocturnal.

Pain intensely severe, and constricting, its chief center being to the back of the mid-sternum and toward the left.

Duration of attack from a few seconds to one or two minutes.

Patient silent and body fixed.

Arterial tension increased as a rule.

Prognosis unfavorable.

## PSEUDO-ANGINA.

The causes are—hysteria, neurasthenia, toxic agents, and reflex irritations.

Occurs at any age (over six years), and usually in females.

Paroxysms arise spontaneously, are periodic and often nocturnal.

Less intense pain, more diffused over the thoracic region: sensation of cardiac distention.

From a half to several hours.

Restlessness and emotional symptoms of causative conditions associated.

Usually not increased.

Entirely favorable.

The vasomotor form of angina must not be confounded with pseudo-angina, which is infinitely more common.

The **prognosis** is bad, yet uncertain. I recall two instances that occurred ten and twelve years ago respectively: the first attacks were survived with no recurrence up to the present. When the arteries are sclerosed (particularly the coronaries) life is often suddenly terminated during the course of the attack. Occasionally the sufferer dies of syncope. The nature of the causal and associated lesions must be considered in estimating the prospect of life. In the vasomotor angina of Nothnägel the outlook is less grave, while in pseudo-angina it is bright.

**Treatment.**—*Prevention* of the attacks in persons who are subject to them is of the most importance. In order to do this all known exciting factors are to be rigidly avoided. The patient should be instructed to carry constantly in a convenient pocket such agents as nitroglycerin and amyl nitrite, beads or *perles* (strength 3 to 5 drops), and also how to use them with judicious care immediately upon the first indication of an approaching paroxysm.

The *treatment of the attack* must be prompt and energetic, though carefully conducted, amyl nitrite being inhaled at once from a handkerchief in doses of 3 to 5 drops according to the severity of the attack. The patient should then be placed in a cool apartment free from disturbing

sounds. *Locally*, the use of the ice-bag may prove efficacious and should be tried at first. Rarely, hot applications (hot cloths or sinapisms) give better results than cold. If the pain is not controlled promptly by this method, the nitrite should be reinforced by the hypodermic injection of morphin (gr.  $\frac{1}{3}$ —0.0216) combined with atropin (gr.  $\frac{1}{120}$ —0.0005). This usually brings speedy relief, and is best suited to those instances in which there is no increase of arterial tension. In cases exhibiting high arterial tension the tincture of nitroglycerin, hypodermically, should be employed (dose  $\mathfrak{m}\text{j}$ —0.066—to be repeated once in a minute if needful).

During the *intervals between the attacks* the aim should be not only to obviate the action of the exciting causes, but also to overcome any predisposing influences that may exist. Schott<sup>1</sup> prefers baths (effervescent) and passive movements to drugs or other methods. In true angina, gymnastic exercises, in the form of passive movements alone, should first be performed by an assistant, but later may be safely entrusted to the patient. In cases in which the arterial tension is habitually exalted, nitroglycerin in increasing doses is to be used perseveringly, beginning with  $\mathfrak{m}\text{j}$  (0.066) and increasing by  $\mathfrak{m}\text{j}$  (0.066) every five or six days until the physiologic effects are produced. Sodium nitrite may be employed similarly, the dose being gr.  $\text{j}$ — $\text{ii}\text{j}$  (0.0648—0.184) three or four times daily. Marked arterial sclerosis, particularly if there be a syphilitic history, is favorably influenced by a long course of potassium iodid. It may be prescribed in doses ranging from gr.  $\text{v}$  to  $\text{xx}$  (0.324—1.296) three times a day. When hypertrophy of the left ventricle is excessive, the use of the following is effective:

Ry. Tr. aconiti rad.,	$\mathfrak{m}\text{xlviij}$ (3.10);
Sodii bromidi,	$\mathfrak{zss}$ (16.0);
Elix. simplicis,	q. s. ad $\mathfrak{z}\text{ii}\text{j}$ (96.0).—M.
Sig. $\mathfrak{z}\text{j}$ (4.0) t. i. d.	

It may be omitted at the end of every two weeks for two or three days. The presence of a gouty diathesis would call for special treatment.

In the *vasomotor* form amyl nitrite and nitroglycerin are most valuable. Additionally, hot foot-baths, followed by friction of the extremities, are also of the highest utility.

The treatment of *pseudo-angina* must be directed at the cause of the complaint—the neurotic condition.

#### IV. CONGENITAL AFFECTIONS OF THE HEART.

THESE result from two leading causes: (1) Arrested development, and (2) Fetal endocarditis. Occasionally, both these factors are operative.

(1) **Arrested development** may produce a great variety of anomalies, some of which may be briefly enumerated: (a) *Acardia*, absence of the organ. (b) *Cor biloculare*, or *reptilian heart*, in which the septum between the auricles and ventricles is absent, thus reducing the number of chambers to two. This is an instance of reversion to a lower type. (c)

<sup>1</sup> *Med. Rec.*, March 11, 1899.

*Absence of the interventricular septum*, the heart consisting of three chambers (*cor trilobulare*). More frequently there is a mere perforation in or an incomplete development of the septum, and this is usually situated in the upper portion. Obstruction of the pulmonary orifice or of the conus arteriosus of the right ventricle are frequently conjoined conditions. (d) *Patency, or incomplete closure of the foramen ovale*. Persistence of the foramen is, in the majority of cases, associated with obstruction of the pulmonary valve, though it may be solitary. (e) An anomaly known as *ectopia cordis* deserves mention. This is a condition in which the sternum is usually divided vertically, and the heart is either entirely exposed or beating just beneath the skin in the cardiac, thoracic, or abdominal region. In this connection another and the most common form of malposition may be added—namely, *dextrocardia*. Here the heart occupies the right side, with reversion of the arch and displacement of the descending aorta to the right of the spinal column. Transposition of other viscera is usually associated. (f) *Anomalies of the valves*.—There may be either a numerical increase or decrease of the cardiac valves, particularly the segments of the semilunar valves of the aortic and pulmonary orifices. Supernumerary segments are usually rudimentary, and at the pulmonary valve one, two, or more are most commonly seen. A decrease in the number of segments is also most frequently observed at the arterial orifices, the aortic and pulmonic semilunar valves then being composed of two segments (bicuspid). This condition may be due to defective development on the one hand, or to endocarditis with resulting cohesion of segments on the other.

(2) **Fetal endocarditis** leads to valvular deficiencies in a manner similar to what occurs during the whole post-natal period. The valve-lesions originating during fetal life are most frequently situated on the right side, probably for the reasons that the ante-natal circulation is more actively carried on in the right than in the left heart, and that the former receives the oxygenated blood from the placenta. They may occur at the pulmonic, the aortic, or the auriculo-ventricular orifices. The changes are of the slow sclerotic form as a rule, and their character is determined largely by the antecedent anomalies that predispose to them. The leaflets present smooth, thickened, and contracted borders. Union of the mitral segments is common, and the chordæ tendineæ are often thickened and contracted. The small rounded bodies that are normally present on the mitral and tricuspid segments (*nodules of Albini*) must not be confounded with pathologic verrucosities.

The most frequent congenital valvular lesion is **stenosis of the pulmonary orifice** as the result of chronic endocarditis. Rarely, it is due directly to defective development, and perhaps more rarely still to endocarditis verrucosa. **Pulmonic constriction** of ante-natal origin may be an associated lesion in other forms of valvular disease in the young adult. With stenosis at the pulmonary orifice, there usually coexist stenosis of the conus arteriosus of the right ventricle, an open foramen ovale, and a patent ductus arteriosus; according to Peacock, “in 86 per cent. of the patients with congenital heart-disease living beyond the twelfth year the lesion is at this orifice.” **Atresia of the pulmonary orifice** occurs, though less frequently than stenosis.

At the **tricuspid orifice** there may be stenosis or contraction of the



valves, producing either obstruction or regurgitation. Similar lesions of the aortic orifice are infrequent. **Congenital mitral disease** also occurs, but only exceptionally; it is then usually associated with tricuspid stenosis. Boys are somewhat more liable to congenital affections of the heart than girls.

**Symptoms.**—There is an almost constant and strikingly distinctive symptom in congenital heart-disease—*cyanosis*. The *tint* of skin observed is variable, being at one time a general duskiness, at another a deep violet, and rarely almost black. This coloration is most noticeable about the lips and mucous membrane of the mouth, the nostrils, conjunctivæ, the fingers, toes, and lobules of the ears, and as a rule is general, though it may be a local condition. The tint may grow less distinct, and even almost vanish, when the child is in perfect repose or sleeping; excitants or efforts at coughing, however, increase the intensity of the discoloration. The cyanotic hue comes on almost invariably during the first week of life. The *fingers* present a decidedly clubbed appearance, and the *nails* are thickened and curved like the claws of certain animals. The *temperature* is subnormal, while the extremities are cool to the feel. *Dyspnea* on exertion and *cough* are usual concomitants. Cyanosis may be due to various causes that may act either singly or more often concurrently. Variot reports two cases with similar cardiac conditions—namely, interventricular perforation, and narrowing of the pulmonary artery, with the aorta arising from both ventricles. Cyanosis was absent from one case, and “this disproves the two leading theories with regard to the origin of cyanosis—the mixture of the two bloods and the obstruction to the pulmonary circulation.”

**Physical Signs.**—In the very young the impulse is feeble (with an absence of a palpable thrill), the *percussion-dulness* is increased, especially to the right, and a loud *systolic murmur* is audible at the pulmonary orifice. When the auriculo-ventricular valves are the seat of endocarditis, the murmur may be apical. In pure pulmonary stenosis the second sound is feeble.

In older children the area of *dulness* is only slightly extended, particularly to the left, while the *murmurs* heard are loud and often musical.

It is interesting to note that in rare instances *cerebral abscess* is an associated condition.

**Differential Diagnosis.**—The distinction between congenital and acquired lesions in children may be assisted by a reference to certain points tabulated below:

CONGENITAL LESIONS.	ACQUIRED LESIONS.
History of almost constant cyanosis, beginning in the first week after birth.	Not so; history of endocarditis or of rheumatism or other complaints in which endocarditis occurs as a complication.
Slight enlargement of the heart. It is of the right ventricle, chiefly non-progressive.	Enlargement marked, frequently involving the left ventricle, and progressive.
Loud and musical murmurs present, audible over upper third of sternum, with small area of transmission upward and to the left; second sound weak.	Audible over apex or base; definite large areas of transmission. Second sound frequently accentuated.
Deficient bodily development.	Bodily development good, as a rule.
Mental faculties in abeyance.	Mental faculties normal.

**Prognosis.**—The prognosis is exceedingly grave. Many succumb within a few days after birth, more than one-half before the expiration of one year, and not less than three-fourths before the end of the third year. Few survive the first decade of life, and fewer still reach full adolescence. Among the forms giving the most favorable prognosis are pulmonary stenosis, especially when of moderate grade, and defective auricular and ventricular septa. In those instances in which life is spared in the first weeks after birth there is a disposition to affections of the lungs (phthisis, hemoptysis), nerve-complaints (convulsions, cerebral hemorrhages).

**Treatment.**—The treatment is, in the main, hygienic. The body must be warmly clad, flannels being worn next the skin, and every source of cold should be carefully guarded against. The diet is to be judiciously arranged, yet liberal, preference being given to the carbohydrates. Gentle exercise when it can be taken is valuable, as are also daily spongings of the surface followed by friction. Special therapeutic indications may arise, and must be met in accordance with general principles, while tonics, such as iron, arsenic, quinin, nux vomica, and cod-liver oil, are frequently applicable.

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## V. DISEASES OF THE ARTERIES.

### ACUTE AORTITIS.

**Pathology.**—The morbid changes coincide with those noted in acute endocarditis, including the ulcerative variety.

**Etiology.**—The causes are not clear, but the condition generally follows the acute infectious diseases (typhoid fever, pneumonia, miliary tuberculosis). Various microorganisms have been discovered to be causal irritants. Boinet and Romary have recently shown that in experimentally produced aortitis a point of lessened resistance (either from traumatism or other previous arterial lesion) is necessary.

**Symptoms.**—The symptoms are *local* and *general*. Of the former, diffuse thoracic *pain* (sometimes severe and throbbing, though more often slight), with more or less substernal *tenderness* under pressure and cardiac *palpitation*, are the chief. Among the *general* symptoms a moderate febrile movement is almost constant. In a certain percentage of cases embolism is betrayed by the usual signs, as rigors, accompanied by a steep temperature-curve. These forms are analogous to the malignant variety of endocarditis. A cardiac murmur may be heard over the base.

**Diagnosis.**—All that the best clinicians can do is to establish a probable diagnosis even in the presence of the most frankly expressed features of the affection. From *acute endocarditis*, aortitis is to be discriminated by its diffuse pain and by the higher seat of its murmur.

The **prognosis** is serious, owing to the liability to infectious emboli and aneurysmal dilatation and the possibility of aortic rupture.

The **treatment** is to be conducted on the same general plan as in acute endocarditis.

## ARTERIAL SCLEROSIS.

(*Arterio-sclerosis; Arterio-capillary Fibrosis; Endarteritis Chronica Deformans; Atheroma.*)

**Definition.**—An overgrowth of the connective tissue of the arterial coats (chiefly and primarily of the intima), followed by calcareous depositions. The elastic tissue of the intima is primarily increased.

**Pathology.**—The most frequent seat of the sclerotic process is the aorta, and the next most common the coronary arteries. Other vessels implicated are the arteries of the brain, the temporals, radials, brachials, ulnars, femorals, and iliaes. On the other hand, certain arteries, as the gastric, hepatic, and mesenteric, are rarely affected. Two forms may be recognized: (*a*) the circumscribed, and (*b*) the diffuse.

(*a*) **Circumscribed Arterio-sclerosis.**—Naturally, the intima presents a smooth internal surface, but when atheromatous changes occur it shows localized areas of thickening. These patchy prominences are often hemispheric in outline, yellowish-white in color, and their favorite seats are the orifices of the branches. They increase in depth and superficial area, and on reaching an advanced stage their interior disintegrates into granular material (*atheromatous abscess*).

In circumscribed or nodular atheroma the microscope discloses the fact that the middle and external coats are the primary seat of the changes, which consist of localized infiltrations. These lesions weaken the media and adventitia, and then (as shown by Thoma) compensatory processes are set up in the intima which lead to the formation of the so-called *atheromatous button*. The latter consists in a hyperplasia of the intima with a deposit of round cells, which causes a gradual increase in thickness; in this way the growing weakness of the middle and outer coats is compensated. When the prominences in the intima undergo softening or liquefaction, rapid dilatation of the affected vessels is apt to occur. The same accident may arise early, or before the intima has reinforced the other layers by its increased thickness and strength.

(*b*) **Diffuse Arterio-sclerosis.**—In this form the morbid process is distributed throughout the greater part of the arterial system, the nodular or circumscribed form being usually combined with it in the aorta. Dilatation of the aorta and of its branches commonly coexist. Apart from the yellowish, translucent, elevated areas, the intima may be smooth and the naked-eye appearances almost normal. The coats, however, and particularly the intima, are very much thickened. Microscopic examination brings to light an extensive proliferation of the subendothelial connective tissue and a hyaline transformation of the entire media, particularly in the larger vessels. The muscular fibers and elastic tissue have in *advanced cases* almost totally disappeared. Necrotic degeneration of the media, especially in the smaller arteries, is also observed, and calcareous deposits, causing rigidity of the walls, occur among the later changes. This is particularly true of the so-called *senile arterio-sclerosis*. Atheromatous abscesses that burst, forming atheromatous ulcers, are likewise common pathologic events in the aged. There may be associated atrophy of the heart, liver, and kidneys, due to a lack of nutritive supply in consequence of the narrowing of the vessels. More commonly, however, in this, and constantly in circumscribed and diffused sclerosis, the heart is moder-



ately hypertrophied. When coronary disease is present, fibrous myocarditis and sclerosis of the aortic cusps may be found associated. The kidneys may also become sclerotic—a condition that will be described hereafter (*vide* Diseases of the Kidneys).

*Sclerosis of the pulmonary artery*, previously referred to in the discussion of the Diseases of the Heart, exhibits all the changes observed in connection with atheroma of the systemic arteries, including aneurysmal dilatation of the trunk and rarely of the main branches. From the terminal tributaries the process may extend to the capillaries, and even to the pulmonary veins (*angio-sclerosis*).

The effect of *arterio-sclerosis* upon the physiologic functions of the vessel-walls, and the pathologic and clinical results are of the utmost importance. The elastic coat is destroyed, and hence the walls cannot bear the blood-pressure as well as in health. This predisposes to dilatation of the vessels (aneurysm).

Another result of extensive atheromatous degeneration of the vessels is an increase in the resistance to the blood-current, and a consequent elevation of the arterial pressure. Furthermore, the loss of elasticity in the coats of the medium-sized and smaller arteries removes an important factor in the propulsion of the blood. The left ventricle, in consequence of this fact, almost invariably becomes hypertrophied in cases of extensive arterio-sclerosis, provided the general nutrition of the patient is still well maintained (Strümpell).

The reduction of the lumen of the vessel, owing to the thickening of the intima, must lessen the blood-supply to the various viscera, and thus in part are explained such secondary affections as fibrous myocarditis, cirrhosis of the kidneys, and cerebral softening.

*Sclerosis of the veins (phlebo-sclerosis)* has rarely been observed as a sequel to arterio-sclerosis. It is, however, not infrequently found in association with hepatic cirrhosis and mitral disease when the portal system and pulmonary veins are involved. It is occasioned by increased intravenous pressure.

Microscopically, thickening of the intima and atrophic degenerative changes in the media are commonly observed. Calcification and hyaline degeneration of the layers also occur, and I have observed them in one of my own cases. Moderate dilatation is not exceptional.

**Etiology.**—The diffuse form has, in part, a special etiology. It may appear in the young, though rarely; I have met with a case in the Medico-Chirurgical Hospital in a man aged twenty-four years. It is, however, most frequent in the middle-aged (who are able-bodied) and in the aged. At an earlier period it occurs as a result of *alcoholism*, *syphilis* (the overshadowing factor), *lead-poisoning*, *gout*, and *chronic nephritis*—agencies that subject the vascular system to undue wear and tear. In old persons atheroma is often *physiologic* and characterizes the natural involution-period of life. *Heredity* may play no inconspicuous part in arterio-sclerosis dependent upon the age. This fact furnishes, to some extent at least, the reason why senile changes in the arteries occur at a much earlier period of life in some instances, and even throughout whole families, than in others. *Negroes* are more liable than *whites* to this form of atheroma, and *males* than *females*, though it is more frequent in the latter sex than the circumscribed variety.

The *general causes* may be thus classified—(1) *Biologic irritants*, as the specific micro-organisms of malaria, rheumatism, and syphilis. (2) *Chemical irritants* (chronic alcoholism, lead-poisoning, uric acid in gout). The above toxic agents produce their effects partly by their direct irritant action, and partly by increasing the resistance in the peripheral vessels and thus raising the arterial pressure. (3) *Bright's Disease*.—It must be admitted that there is a class of cases in which arterio-sclerosis is secondary to Bright's disease, but when found in association the former is more frequently the primary disease than the latter. The chief causes of chronic nephritis are also capable of setting up atheroma, and hence it must not be forgotten that the two diseases may develop independently of one another, and yet simultaneously, in consequence of the action of a common cause. (4) *Constant overfilling of the blood-vessels*, resulting from excesses in eating and drinking, in the opinion of certain authors, also causes arterio-sclerosis. (5) *Muscular over-strain*, which augments the blood-pressure while at the same time obstructing the peripheral circulation, is a leading factor. (6) The main causes of sclerosis of the pulmonary artery are *mitral disease* and *emphysema*.

**Clinical History.**—The disease may exist for years without becoming apparent; or it may be discovered incidentally at *autopsy* while palpating the arteries during the course of an examination for some supposed local visceral affection. The accessible peripheral vessels (radial, temporal, femoral, and brachial) should be carefully felt when the presence of the disease is suspected. The *walls* of the affected artery feel *hard*, and the *pulse*, owing to increased tension, is incompressible; as a result of this rigidity of the arterial walls the degree of vascular tension is difficult of estimation. In marked cases the sensation is similar to that perceived when grasping a goose's neck, and in such instances the pulse-wave may not be detectable on palpation. Again, the *tension* may be high, and yet sclerosis of the vessel-wall be slight or absent. When doubt arises as to whether or not sclerosis exists, the pulse should be palpated by means of two fingers. If now, while compression of the pulse is made with the index-finger, the middle-finger detects a pulse-wave, arterio-sclerosis is present. On account of the loss of elasticity of the vascular walls the pulse is retarded, and the *sphygmogram* shows a short sloping ascent, a wide top, and a slow, gradual descent, with almost an effacement of the dicrotic notch.

The opposition of the increased resistance to the circulating medium (due to the rigid vessel-wall) in the outlying portions of the body calls forth a correspondingly increased cardiac action, and thus *hypertrophy of the left ventricle* is engendered, with its customary symptoms and physical signs, including the ringing, accentuated second sound. The balance of the cardio-vascular forces may thus be maintained for a long period of time, during which the health of the patient often remains unimpaired. It happens sometimes that hypertrophy preponderates and veils completely the symptoms of arterio-sclerosis. In elderly persons suffering from atheroma the first sound is often surprisingly feeble. *Myocardial degenerations* frequently come on in the later stages, when dilatation of the left ventricle, accompanied by a mitral systolic murmur and marked rapidity of the pulse, may supervene. The *aorta* may be so dilated as to give rise to an abnormal area of dulness in the upper sternal region.

*Palpitation, dyspnea on exertion*, a feeling of *precordial constriction*, and *light febrile attacks* are not uncommon. *Angina pectoris* is an infrequent symptom except in coronary atheroma.

It cannot be stated absolutely that involvement of the *external arteries* implies a serious involvement of the aorta and its main branches. On the other hand, the circumscribed variety is not attended with characteristic alteration of the pulse. The *pathologic*, and more particularly the *clinical*, events above described may be more pronounced at one portion of the body than at others, and this fact has given rise to several distinct or *special types* (apart from the general or cardio-vascular form first depicted) according to the seat of the most marked symptoms and lesions. Among the latter I would mention the (a) cerebral, (b) pulmonary, (c) renal, and (d) peripheral types.

(a) **Cerebral Type.**—In the milder grades of this type such symptoms as headache, tinnitus, vertigo, syncopal attacks, and local palsies are variously blended as a rule. I have had under my care for two years a case of marked arterio-sclerosis in a man aged eighty years in whom tinnitus aurium and vertigo, with mild melancholia, are the only constant symptoms; on two occasions temporary aphasia was superadded.

Especially in the aged, the condition is apt to lead to *thrombosis* or *cerebral embolism*, small emboli being detached from the aortic area and conveyed to the brain, with the development subsequently of the symptoms of anemic softening of the latter. The loss of elasticity of the vessel-walls in atheroma renders them more liable to rupture than normal arteries, while the tension is much increased. Under these circumstances the danger from apoplexy is quite obvious.

(b) **Pulmonary atheroma** is considered in its clinical relations in connection with the diseases of the heart and lungs.

(c) The **renal type** includes those instances of kidney-lesion that are associated with or follow general arterio-sclerosis. The condition is essentially an atrophic nephritis, due to the diminution of the blood-supply to the organs in consequence of the narrowed lumen of the renal arteries.

(d) In the **peripheral type** the arteries leading to the extremities become obliterated to such an extent as to cause starvation of the tissue, with resulting gangrene.

**Diagnosis.**—Hardened arteries, increased arterial tension, left ventricular hypertrophy, and marked accentuation of the aortic second sound form a grouping of clinical characters that leaves no doubt as to the diagnosis. Not infrequently it is the occurrence of apoplexy, acute cardiac dilatation, or of some other such accident that leads to the discovery of general arterial sclerosis.

Raw has successfully skiagraphed the arteries by a special method; and C. Beck<sup>1</sup> and others have found that the *x-rays* are useful in determining the extent of arterio-sclerosis (*e. g.* whether local or general).

To **differentiate** the murmurs of dilatation of the left ventricle following the hypertrophy of this disease from *organic valvular lesions* is only possible by the history or the results of treatment. The systolic murmur over the aortic area in atheroma may suggest *aortic stenosis*. In such cases, however, the second sound is loud, and the pulse more voluminous than in aortic constriction (*vide* Aortic Stenosis).

<sup>1</sup> *N. Y. Med. Journ.*, Jan. 22, 1898.



**Prognosis.**—Arterio-capillary fibrosis is an exceedingly chronic, though usually a progressive disease, and frequently it terminates life. The axiom that a man is as old as his arteries has been borne out by the test of extensive clinical observation. The condition may prove fatal either with great suddenness, as when it occasions apoplexy, or with unwonted slowness. Very rarely the aorta ruptures at the seat of an atheromatous ulcer, causing instant death.

**Treatment.**—Though the progress of the disease cannot in most instances be successfully stayed, it can be retarded frequently by correcting aggravating habits and by removing the influence of ascertainable causes. The syphilitic taint, if present, requires the liberal use of the iodids.

The *diet* must be simple and free from stimulating properties; skimmed milk is excellent, particularly if renal symptoms be manifested. Perhaps no other agent is so generally serviceable in all cases, especially in the earlier stages, as potassium iodid, which should be administered for several years, combined with appropriate gymnastics to regulate the bodily functions.

For the increased arterial tension nitroglycerin or the other nitrites should be employed. The former should be given in increasing doses until an impression has been made upon the blood-pressure, after which this effect should merely be maintained.

For the local aortic symptoms (fever, pain) absolute rest, a liquid and unirritating diet, and a small blister are most efficacious, together with internal minute doses of calomel, quinin, and potassium iodid.

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## ANEURYSM.

**Definition.**—A true aneurysm is a circumscribed dilatation of an artery, formed of one or more of its coats.

Classified according to their form, aneurysms are—(1) sacculated, (2) cylindric, and (3) fusiform. They are termed *axial* when the complete circumference of the vessel participates in this dilatation, and *peripheral* when a single sac is confined to the side of the vascular duct.

*Miliary aneurysms* occur along the course of the cerebral vessels, and are so termed on account of their minute size. On the other hand, they may attain the size of the human skull.

By a *false aneurysm* is meant one in which the coats are ruptured and the blood is found in the adjacent tissues.

A *dissecting aneurysm* is one that, owing to laceration of the internal coat, dissects between the layers of the vessel-wall. For its *seat* it usually selects the aorta, and may traverse its entire length.

An *arterio-venous aneurysm* arises from a direct fistulous connection between an artery and a vein (*aneurysmal varix*), or an aneurysmal sac may intervene (*varicose aneurysm*).

**Pathology and Pathogenesis.**—The wall of the aneurysm is commonly the seat of arterio-sclerosis, which, according to Malkoff,<sup>1</sup> is a compensatory arrangement. The intima is thickened, and to a less extent the media in the early stages. The media probably weakens first in most cases, and extreme atrophy of both the intima and media is not uncommon in the later stages, so that the wall of the sac is often formed

<sup>1</sup> Ziegler's *Beiträge*, 1899, xxv.

almost exclusively of the adventitia. The intima (as in Daland's case of aortic aneurysm, in which there were both an old and a new transverse rent) may become lacerated, and finally the media and adventitia tear; this results in rupture unless the adherent neighboring structures compensate for the natural wall.

The blood in the aneurysmal sac is partly fluid and is composed of old and new thrombi. The latter when comparatively recent may be soft, and when old may be firm or even calcified, yellowish in color, and adherent to the wall. With the progressive enlargement of the aneurysm surrounding organs are apt to be compressed and their functions disturbed.

**Etiology.**—Among recognized *causes* are—(1) **Arterio-sclerosis.**—It follows that the same conditions that originate the latter must also tend to bring about aneurysms. Chief among these is syphilis. According to Rasch, syphilis was present in 56 per cent. and probably in 82 per cent. of 25 aneurysms of the aorta discovered in the course of 3165 necropsies at Copenhagen. (2) **Sudden Great Strain.**—This may be productive of aneurysm, if some previous local impairment of the arterial walls have been present, however slight. In this way only can the fact be accounted for that most instances of aneurysm occur during the period of greatest bodily activity in the male sex. (3) **Embotic plugging of a vessel,** if complete, may cause aneurysmal dilatation on the proximal side of the point of obstruction. The development of aneurysm may under these circumstances be facilitated by the mechanical effects of the embolus, which may be of calcareous hardness, as when it comes from diseased heart-valves. Infectious emboli set up inflammation and softening. (4) **Mycotic Aneurysms.**—That aneurysms sometimes owe their existence to mycotic origin was first pointed out by Osler, who found an abundant growth of micrococci in the aneurysmal sacs. They are met with in ulcerative endocarditis, and are often small and usually multiple. (5) **Traumatism.**—Aneurysms have been produced experimentally by traumatism (Malkoff); hence it is obvious that it may become one of the assignable causes. (6) **Age and Sex.**—Aneurysms are most frequent between the *thirtieth* and *fiftieth* years, this being the period of greatest physical exertion. The *male sex* is more frequently affected than the *female*, owing to differences in occupation.

#### ANEURYSM OF THE THORACIC AORTA.

(*Aneurysma Aortæ.*)

The *thoracic portion* of the aorta is involved in about 75 per cent. of the cases, and the *abdominal aorta* and its branches furnish about 25 per cent. Within the thorax nearly 60 per cent. of the cases originate in the *ascending portion of the aorta*, while nearly 30 per cent. are seated upon its *arch* (Lyman).

**Symptoms.**—Intrathoracic aneurysms may exist, particularly if they are small, without symptoms or noticeable physical signs. When they attain to any considerable dimensions, however, they usually excite characteristic signs and distressing symptoms, the latter being the results of direct pressure, and hence varying with the seat and direction of the progressive enlargement. In a few instances truly diagnostic symptoms are present in the absence of a detectable tumor or physical signs. Finally, the more characteristic features—the tumor inclusive—may be

more or less nearly intermittent. It is important to note the condition of the neighboring organs upon which pressure is exerted by the growing aneurysm, as well as the symptoms and signs thus occasioned. Aneurysms of the **ascending portion** of the arch usually *compress* the vena cava, causing *distention of the veins* of the head and arms, though in a proportionately small number of cases the subclavian may be the only vein compressed, with resulting *enlargement* and *edema* of the right arm. The largest aneurysms may even compress the inferior vena cava, causing edema of the lower extremities. The *heart* is displaced outward toward the left pleura, usually forward and upward, and rarely causing erosion of the ribs and sternum. The right recurrent laryngeal nerve may be implicated, giving rise to *dyspnea* and *aphonia*. *Pain* is a constant feature. When a *tumor* appears, it occupies the upper two or three intercostal spaces near the right border of the sternum.

Aneurysms of the **transverse portion** of the aorta, when they attain any considerable size, cause the most intense symptoms, owing to the relatively shorter antero-posterior diameter of the chest at this point, in consequence of which greater *compression* of the neighboring tissues takes place. By protruding backward they may exert pressure upon the trachea, causing *paroxysmal cough* and *dyspnea*, or on the esophagus, causing *dysphagia*; these are common events. The pressure may fall also upon the bronchus, inducing *dyspnea*, *bronchorrhea*, and *dilatation*, the latter in turn sometimes leading to circumscribed abscess. The left recurrent laryngeal nerve may be implicated, with resulting *aphonia*.

*Upward extension* of the aneurysmal process, with involvement of the coats of the carotid and subclavian on the left side, or of the innominate and carotid on the right, may occur. The *sympathetic nerves* in the cervical region may be irritated, causing dilatation; or they may be paralyzed, causing contraction of the *pupils*. Compression of the *thoracic duct* may occur, with resulting *rapid emaciation*. A *tumor* may appear in the jugular fossa.

The aneurysm may grow *forward*, in which event it lies directly behind the manubrium, which from the pressure becomes eroded and may finally disappear in part. In aneurysms involving the transverse portion of the arch, lateral pressure, both toward the right and the left, is also made, causing recession and compression of the lungs.

When the **descending portion** of the arch is affected the pressure is exerted upon the spinal column to the right, and upon the tissues as far as the shoulder-blade to the left. As a consequence of destruction and absorption of the vertebræ, compression of the spinal cord may ensue, and is an intensely painful process. Pressure may be made upon the esophagus, causing *dysphagia*, or upon the left bronchus, causing *bronchiectasis*, with its usual *sequelæ* (bronchorrhea, fetid bronchitis, gangrene of the lung).

The sac may, in consequence of the slow ulcerative process that attends its progress, *rupture* eventually into the bronchus, the pericardium (when situated near the sinuses of Valsalva), the pleura, the superior vena cava, or the esophagus, with sudden death as the result. Frequently repeated small *hemorrhages*, due to weepings from the thinned walls, may precede the fatal rupture. I saw a case of aneurysm of the transverse portion in which rupture into the esophagus resulted, with instantaneous death.

When the tumor has reached the subcutaneous tissue and bulges ex-



ternally, the skin covering it becomes tense and shining, and with increased pressure the surface becomes reddened and finally necrotic. The necrosed area is covered with a dry brown scab, which later is thrown off, leaving an oozing surface. Rupture soon follows.

**Leading Symptoms in Detail.**—Among these *pain* stands primarily, being the first and most constant. It is of two kinds: (*a*) due to direct pressure upon and stretching of the nerves. When aneurysm is developed suddenly, a sharp, excruciating pain is felt in the upper sternal region, accompanied by a feeling of “something giving way.” In consequence of the stretching of the nerves a constant pain is experienced that is subject to exacerbations when the intra-aneurysmal pressure is raised. Pressure against the bony structures causes erosion, and usually produces a continuous boring pain. In a recent case of aneurysm shown in clinic at the Medico-Chirurgical College, however, a tumor that had eroded the right half of the sternum, and of the size of a goose’s egg, had given rise to no suffering whatever. In latent aneurysm there is an absence of pain until the growth terminates life. Anginose attacks sometimes occur when the sac has its seat near to the heart. (*b*) Reflected pains of a neuralgic character may be excited by aneurysm. This is true, in particular, of aneurysms situated in the transverse portion of the aorta, in which instances pain is commonly felt in the region of the neck and occiput and down the left arm. When the growth is situated along the course of the descending aorta, intercostal neuralgia may be excited, and it is probable that pain of this sort is due to pressure upon the nerve-trunks.

*Cough.*—The cough is paroxysmal, and frequently has a peculiar brazen, ringing character that points to its laryngeal seat. Pressure upon the windpipe excites a paroxysmal dry cough. Compression of a bronchus may lead to bronchiectasis, and the cough then occurs only in long and severe paroxysms which recur at intervals of a day or even longer, and are attended with copious, thick, ropy expectoration (*vide* Bronchiectasis).

*Dyspnea* is a conspicuous symptom in aneurysm of the transverse portion of the aorta (the aneurysm of symptoms—Broadbent). It arises (*a*) most frequently in consequence of pressure upon the recurrent laryngeal nerve, (*b*) direct pressure on the trachea, and (*c*) from pressure on the left bronchus. Marked stridor may accompany the first variety.

*Paralysis* of the vocal bands is occasioned by compression of the recurrent laryngeals, particularly the left, while a slight degree of compression or irritation of the same nerve causes *spasm* of the vocal cords. The symptoms indicating the presence of these conditions are hoarseness, cough, and aphonia respectively. The laryngoscope should be employed, since paralysis of one of the abductors may be present without giving rise to appreciable symptoms.

*Hemorrhage* may occur as a slow oozing, either from the point of compression in the trachea or externally; in either case the amount of blood lost is small. Profuse bleedings (often producing sudden death) take place in consequence of rupture of the sac into the lung, the bronchus, or the trachea.

*Deglutition* may be difficult, owing to compression of the esophagus. When an aneurysm has been diagnosticated or even suspected, the esoph-

ageal sound should not be passed for purposes of exploration, on account of danger of rupture of the sac.

*Compression and irritation of the sympathetic system of nerves* cause pupillary changes that have already been mentioned. With dilatation of the pupil there may be observed pallor of one side of the face, due to stimulation of the vaso-dilator fibers; on the other hand, with contraction of the pupil (due to paralysis of the dilator fibers) there is hyperemia of one side of the face and unilateral sweating.

*Clubbing of the fingers and incurvation of the nails* are not rarely met with in thoracic aneurysm.

**Physical Signs.**—*Inspection.*—Visible pulsation is one of the earliest appreciable signs. It is most frequently observed at the right side of the sternum, above the level of the third rib (second interspace), and much less frequently on the left side over a corresponding area. In aneurysm of the transverse portion pulsation may be seen at the episternal notch, though an impulse here may also be due to nervous palpitation, and have no connection with aneurysmal growths. The pulsation may occur in the absence of the slightest bulging; when associated with swelling, however, its diagnostic value is infinitely greater.

Involvement of the innominate artery produces pulsation in the neck above the sterno-clavicular junction, or less commonly above the sternum. Corresponding to the site of visible impulse, there is, sooner or later, bulging in most instances. It may, however, be so slight as to elude detection unless the keenest observation be practised, and in not a few instances the tumor itself is invisible from the front of the body, but is recognizable looking from behind or from either side. Again, on allowing the light to fall obliquely upon the chest slight prominences may be brought to view that would otherwise be inappreciable.

When the aneurysm is situated in the ascending part of the arch, the most frequent seat of the bulging—which varies in size from a hen's egg to a cocoanut—is over the first and second right interspaces near to, and frequently involving, a portion of the sternum; when seated just beyond the aortic orifice, a pulsating prominence may occupy the third interspace along the left sternal border; situated in the transverse section of the aorta, bulging of the upper part of the sternum is a frequent event, or a projection into the cervical fossa may occur, though with comparative rarity. In the descending portion the swelling, when present, is in the second and third left interspaces near to the sternum, or very rarely in the left scapular zone. The skin over the tumors has been described. The apex-beat is displaced downward and outward, chiefly from pressure, though to a lesser extent also from hypertrophy (functional).

*Palpation.*—The protrusion presents a more or less yielding and elastic mass, and when superficially seated fluctuation may be obtainable. The degree, and the rhythmic expansile character of the pulsation are to be noted, and also the fact that there is an alternate contraction and dilatation of the sac in every direction—a distinctive feature.

If the aneurysm is largely concealed, bimanual palpation should be employed, the palm of one hand being placed over the spine and that of the other over the sternum. In an inconsiderable number of cases aneurysmal pulsation is only yielded when the finger-tips are used, and quite rarely only at the end of expiration. A diastolic shock is often

perceived, and forms a physical sign of no little value. A distinct systolic shock, sometimes accompanied by a purring fremitus, can also be felt over the aneurysmal sac.

*Percussion.*—If the growth be deep-seated, percussion may give negative results; when, however, the tumor causes bulging or comes in contact with the chest-wall, a proportionate area of flatness is presented. The abnormal field of dulness may be the only symptom present, as in an instance of suspected aneurysm that recently came under my care. Aneurysms of the ascending arch give flatness to the right of the sternum; those of the transverse arch, over the upper part of the sternum and to the left; while those of the descending portion are revealed by a flat area between the spine and the left scapula. With flatness of the percussion-note there is a sense of increased resistance. There is generally a moderate increase in the area of cardiac dulness. Conversely the left ventricle has been found of diminished size at necropsy.

*Auscultatory percussion* (practised after the method of Sansom and Ewart) quite often gives valuable results.

*Auscultation.*—Since murmurs owe their origin, in great part, to the presence of fibrin in the sac, they may often be absent, and this even in the case of large aneurysms. When, as is usual, a murmur is present, it is systolic in rhythm, heard with greatest intensity over the flat area or body of the tumor, and is transmitted in the direction of the blood-stream, being, therefore, distinctly audible in the vessels of the neck and along the course of the aorta. The murmur has a booming quality.

Aortic regurgitation may be considered as associated with aneurysm near the aortic ring when a double murmur is heard. In a few instances the diastolic bruit is alone detectable. A much intensified, ringing second sound is present (unless marked aortic regurgitation coexists), and is a sign of the utmost significance for diagnosis.

**The Peripheral Arteries.**—The pulse in the vessels beyond the aneurysm is slowed. Hence the two radial pulses may exhibit differences in time. The volume of the pulse beyond the aneurysm is also lessened, and in cases

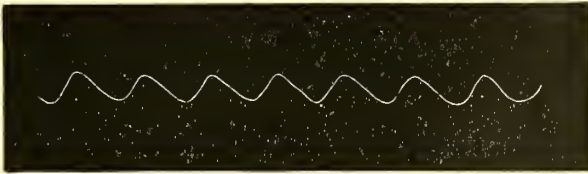


FIG. 57.—Sphygmogram of a case of aneurysm of the left subclavian artery (Foster).

of aneurysm of the abdominal aorta or the femorals it may be obliterated. Such differences as these will not only point to the existence of thoracic aneurysm, but also may indicate its seat. Thus, if there be dilatation of the transverse arch with no implication of the innominate, the pulse at the right wrist is strong and almost simultaneous with the cardiac impulse, while that on the left side is small, weak, and more retarded. If the reverse be true, then the aneurysm may be near to or involve the innominate.

The sphygmogram exhibits a slanting up-stroke with obliteration of the secondary wave (Fig. 57), though its characters are by no means constant.



**Tracheal Tugging.**—This sign may be practised while the patient is sitting or standing with the chin slightly elevated. The cricoid cartilage is then grasped between the thumb and forefinger and pushed gently upward so as to stretch the trachea. The patient must cease breathing momentarily, when, if this sign be present, there will be a downward dragging or tugging at each systole. The transmitted pulsations from the cervical vessels must not be confounded with the up-and-down movement of the trachea.

A new method of eliciting tracheal tugging, first suggested by Ewart, has been pretty widely adopted, and possesses the advantage of ensuring greater delicacy of touch than the old. He stands behind the patient, supporting the head of the latter against his body, and the cricoid is then grasped firmly between the tips of the forefingers. The method is in other respects similar to that previously described.

As shown by Toulmin, tracheal tugging may be present in health and in other diseases. The sign is probably one of less value than has been supposed.

**Diagnosis.**—In the presence of the following points the existence of thoracic aneurysm may be confidently inferred: (1) Antecedent arteriosclerosis (with the appropriate causes of the latter); (2) History of other etiologic factors, as age (between thirty and forty-five years) and occupation (such as entail unusual muscular strain); (3) Pressure-symptoms, as pain, dyspnea, aphonia, cough (either laryngeal or bronchial), bronchorrhea, dysphagia, edema, vasomotor disturbances; (4) Physical signs of a pulsating tumor (including the abnormal area of dulness, systolic murmurs, the systolic and diastolic shock, and tracheal tugging) somewhere along the course of the arch or its great branches, with or without differences in the volume and time of radial or carotid pulses. There are, however, several classes of cases which offer difficulties that are sometimes insurmountable: (*a*) Those in which the aneurysm is small and deep-seated. Here the symptoms and physical signs are indefinite. There may be thoracic oppression, in which pain may radiate to the left shoulder, and mild pressure-symptoms—a group of suspicious features merely—sometimes appear. I have under my observation now a patient suffering from aneurysm of the ascending aorta in which for a long time left-sided intercostal neuralgia was the only symptom. (*b*) Aneurysm of the transverse arch, in which the pressure-symptoms are more or less pronounced, but with no physical signs. In such, a clear history suffices to complete the diagnosis. Pressure-symptoms, on the other hand, without etiologic factors are just as likely to be due to other causes. (*c*) Those cases in which the more characteristic features are manifested intermittently. Fortunately, a proper diagnosis of aneurysm in obscure cases can be often made by the aid of the *x*-rays.

Extremely obscure are many of the cases in which the only symptoms manifested point to irritation of the trachea or bronchial tubes, with paroxysmal cough, bronchorrhea, and sometimes the signs of bronchiectasis. In a recent case of this sort tracheoscopic examination revealed compression of the windpipe, making clear the nature of the affection. In still another instance, in which laryngeal dyspnea and cough, with bronchorrhea, were the only symptoms, a laryngoscopic examination determined the diagnosis, in that it brought to view bilateral paralysis of the abduc-

tors of the vocal bands. Systolic pulsation of the larynx and trachea (Oliver's sign) is of considerable value when present, especially in otherwise dubious cases.

**Differential Diagnosis.**—The affections from which intrathoracic aneurysm must be distinguished are *pulsating empyema*, *pulmonary tuberculosis*, *abnormal pulsation of the aorta*, and *solid tumors*. Of the latter, those simulating aneurysm are carcinoma, sarcoma, and enlarged lymph-glands. These *mediastinal tumors* may duplicate all of the pressure-symptoms, though they are less apt to cause bulging, and less apt still to excite abnormal pulsation; when pulsation is noted it is observed to be quick, and not deliberate, heaving, and expansile, as in aneurysm. Solid growths also lack the characteristic shock—both systolic and diastolic—of aneurysm. The cardio-vascular symptoms are usually wanting in the case of solid tumors, especially the moderate hypertrophy, accentuation of the second sound, tracheal tugging, and the difference between the radial pulses.

*Carcinoma of the mediastinum* usually gives a history of the disease in other parts of the body, with enlargement of the axillary or other superficial lymphatic structures, and later the characteristic cachexia, this being particularly marked in carcinoma of the esophagus.

*Abnormal pulsation in the aorta* is noted in neurotic subjects, mostly females, and in aortic regurgitation; less frequently it is associated with retraction of the right lung, with spinal curvature, and with displacement of the aorta. In the case of the latter two conditions a careful consideration of the causal states and the absence of the characteristic physical signs would lead to a correct diagnosis. *Aortic regurgitation* is frequently associated with aneurysm of the arch, and in its course there is developed, not infrequently, a dilatation of the ascending portion of the aorta. The diagnosis of aneurysm of the arch of the aorta should not be made, even when all the characteristic physical signs of aortic regurgitation are present in any given case, unless the signs of the pulsating tumor above the heart be unmistakable. Dynamic pulsation of a neurotic origin is seen and felt in the episternal notch, as a rule, and a correct appreciation of the nervous element, whether inherited or acquired, will prevent the observer from committing an error.

*Pulsating empyema* can only be confounded with large aneurysmal growths, and, as pointed out by Wilson, it does not have the same definite relation to the central long axis of the body as do aneurysms. The abnormal area of dullness is situated at the base of the lung in empyema, and usually extends over a larger superficial area or is less circumscribed than in aneurysm. In empyema, moreover, the pulsation is not expansile, but is caused by pressure on the respiratory movements. Auscultation in empyema renders audible neither a bruit nor the double shock of aneurysm; the pressure-symptoms and pulse-characters are also entirely wanting.

*Pulmonary tuberculosis* may be mistaken for thoracic aneurysm. When an aneurysm compresses a bronchus, bronchiectasis, attended with cough, bronchorrhea, fever, and emaciation, may be the result; but in phthisis the fever and emaciation are more pronounced, tubercle bacilli are present, whilst the characteristic cardio-vascular signs of aneurysm are absent.

**Prognosis.**—The occurrence of perforation and consequent speedy death in unsuspected cases must be recollected. In other instances the end is approached in a very gradual manner, and cases in which rupture does not supervene sometimes pursue the general course of chronic valvular affections of the heart. The condition ends in death as a rule, and the *immediate causes* of the fatal issue are as follows: (1) Rupture of the aneurysm, followed by hemorrhage into any of the adjacent cavities or organs (pericardium, heart, large vessels, mediastinum, trachea, a bronchus, esophagus, lungs, pleura, spinal canal); it may, though rarely, rupture externally, in which case slight hemorrhages occur and life may last for weeks; (2) Gradual asthenia; (3) Direct pressure; (4) Independent diseases, either primary or secondary to, and induced by, the aneurysm. Among these *pulmonary complications*—fibrinous pneumonia, abscess, gangrene, tuberculosis—are of first importance.

**Treatment.**—There are two objects of treatment around which all others center in the management of this disease—first, the promotion of coagulation of the blood, and secondly, the contraction of the sac. The **clotting of the blood** within the growth may be greatly favored by retarding the blood-current. Nothing so well accomplishes this object as *absolute rest* in the recumbent posture. This cannot always be rigidly enforced, but muscular exertion must be minimized, and mental application and emotional excitement avoided; stimulants, arterial and nervous, are to be eschewed for like reasons. Palpitation of the heart, when present, is to be allayed by the local use of the ice-bag. The coagulability of the blood is also increased by removing as far as possible the liquid portion of the diet.

The measures already indicated tend to lessen the volume of blood and the intra-aneurysmal pressure, thus inviting **contraction of the sac** as well as consolidation of its contents. Among medicinal agents, ergot and potassium iodid have been employed, the latter with good effects. The exact manner in which the iodid produces its favorable results in these cases is unknown, though most probably it acts upon the vascular walls, and hence would be most efficacious when the disease is of syphilitic origin; this view accords with my own personal experience. I would advise against the internal use of ergot, which can have little to recommend it, and the prolonged use of which may be attended with unpleasant effects. Langenbeck and others have obtained good results from the direct injection into the sac of the aqueous extract of ergotin dissolved in water or glycerin, every day or two. When employed in this manner ergotin induces contraction of the smooth muscles in the wall of the aneurysm. Numerous observers have resorted to the use of horse-hair, fine wire, fine catgut, slender watch-springs, with a view to coagulating the blood as it comes in contact with these foreign bodies. Electrolysis is a method that has been warmly advocated (Lorcta).

Combined wiring and electrolysis (Corradi's method) has been successfully employed by Rosenstein, Kerr, D. D. Stewart, and Herchy. The details of the method are briefly as follows:

A piece of fine, slender wire, several feet in length, is passed directly from a spool through a hypodermic needle, so that the wire curls up within. This is now attached to the positive pole, while the negative is connected with a surface pad placed over the abdomen or with an insu-



lated needle inserted into the sac, and the current is then passed through. It is important to test the strength of the current beforehand by inserting the needle attached to the positive pole into the white of an egg and observing its power to coagulate albumin. Each application of the current should last from one to two hours. It is not, however, without serious dangers (hemorrhage and embolism).

After the same method galvano-puncture has long been resorted to, and in the hands of some clinicians with encouraging results. The cases that receive most benefit from the above measures belong to the saccular variety; this is also true of the special plan first commended by Tufnell, which is especially applicable in the earlier stages. Tufnell's method is founded upon two main principles—absolute rest in the recumbent posture, and a much-restricted, dry diet. With physical rest a quiet mental state should be conjoined. The diet is as follows: Breakfast, 2 ounces (64.0) of bread and butter and 2 ounces (64.0) of milk; for dinner, 2 or 3 ounces (64.0–96.0) of meat and 3 or 4 ounces (96.0–128.0) of milk or claret; for supper, 2 ounces (64.0) of bread and 2 ounces (64.0) of milk.

The chief advantages growing out of this method are the lessened number and decreased force of the heart-beats in consequence of the posture and bodily rest, and the diminution of the blood-volume in consequence of the dietetic restrictions. It should be persevered in for several months. His bowels should be regulated, and he should be told not to strain while at stool.

A. E. Wright has particularly insisted upon the value of calcium salts in increasing the coagulability of the blood (gr. x to xv—0.648 to 0.972, t. i. d., may be given). A. E. Taylor's studies show that if it is desired to saturate the body with calcium salts, water should be given in abundance.

Injections of gelatin in aneurysm have a specially favorable effect according to certain observers. Moyer<sup>1</sup> reviews the literature, and in the main his conclusions are: Gelatin solutions are of some value in the treatment of saccular aneurysms, but not of the diffused forms. Solutions not stronger than 1 per cent. should be used; they should be kept in a brood-oven to determine bacterial growth, and great care should be taken in the technique. Absolute rest in bed should be enjoined. This method is worthy of extended trial, but great caution and watchfulness must be exercised in its administration.

*Special Symptoms.*—Pain is often relieved by potassium iodid. When marked arterial sclerosis is present I have seen relief from pain afforded by the internal use of nitroglycerin (mj to ij—0.066 to 0.133, three or four times a day). In the later stages morphin should be given to allay suffering. When there is bulging the pain may be assuaged by the local use of the ice-bag or by a belladonna plaster.

Dyspnea and great venous congestion are to be met by free bleedings from a vein, and tracheotomy may be required if the dyspnea be shown to be due to bilateral paralysis of the abductors. In dyspnea arising from pressure on the trachea or bronchus, however, tracheotomy would be a valueless expedient. When the aneurysm forms a large external tumor the application of an elastic bandage to the chest may be both agreeable and advantageous, as in a case referred to by Osler.

<sup>1</sup> *Medicine*, March, 1899.

## ANEURYSM OF THE ABDOMINAL AORTA.

The vicinity of the celiac axis is the favorite seat of abdominal aneurysm, which is by no means so common a condition as intrathoracic aneurysm, though not rare. It may assume a fusiform or saccular nature.

**Symptoms.**—The tumor may grow backward; but more frequently its growth is in a forward direction. Projecting from the *posterior wall*, it usually erodes the vertebrae, and compression of the cord is apt to take place, producing *paraplegia*, preceded by *tingling* and *numbness* of the legs.

*Pain* is the leading symptom. It may be neuralgic or of a boring or gnawing character, due to destruction of the bone. Rarely, the aneurysm perforates the diaphragm, and finally *ruptures* into the lungs or pleura. Arising from the *anterior wall*, it may early form a well-defined tumor. It may, however, when situated high up or near the diaphragm, conceal itself until it has attained a comparatively large size, as in a case recently under my care at the Medico-Chirurgical Hospital. *Vomiting* and *gastralgic seizures* may be troublesome, and the fact that *embolism* of the superior mesenteric artery may occur and give rise to severe colicky pains must be recollected. *Jaundice* has been observed.

**Physical Signs.**—Epigastric pulsation may be *visible*, and occasionally an epigastric swelling. The *palpating* hand detects a heaving, expansile pulsation that may be accompanied by a thrill. When the tumor hugs the diaphragm the pulsation may be double. The femoral pulse is diminished in volume and delayed. An abnormal area of dullness may be present. In most instances a *soft bruit* is audible. The diastolic murmur and shock of *intrathoracic aneurysm* are quite usually absent.

**Diagnosis.**—A certain diagnosis demands the presence of a definite growth that is seizable and has a heaving, expansile pulsation. Mere pulsation attended with a thrill and a systolic murmur are insufficient.

**Differential Diagnosis.**—A *throbbing aorta*, as met with in neurotic females and in anemia (particularly in instances of the traumatic form), is not infrequently distinguished from aneurysm of the abdominal aorta with great difficulty. It does not, however, present a tumor that can be held in the grasp and possessing an expansile pulsation, as in aneurysm.

When *solid growths* lie upon the aorta the latter may manifest pulsation, a thrill, and a systolic murmur, but the very general absence of pulsation, owing to the fact that the tumor falls forward when the patient is placed in the knee-elbow position, suffices usually to differentiate the condition from genuine aneurysm. Again, expansile pulsation is not evinced by a solid growth.

The **prognosis** is very gloomy. Very rarely, however, nature effects a cure if the conditions be favorable. "Death may result from (a) the complete obliteration of the lumen by clots; (b) compression-paraplegia; (c) rupture either into the pleura, retroperitoneal tissues, peritoneum, the intestines, or, very commonly, into the duodenum; (d) embolism of the superior mesenteric artery, producing infarction of the intestines" (Osler).

**Treatment.**—Apart from the measures indicated for thoracic aneurysm, there is one means of cure that may be tried if the growth be low down—viz. *pressure*. This must be maintained for twenty-four hours at least under an anesthetic. It is best to make steady pressure on the proximal portion of the vessel, and unless practised with great care the sac will be damaged and death ensue.

#### ANEURYSM OF THE PULMONARY ARTERY.

Dilatation of the pulmonary artery is of frequent occurrence in affections that oppose obstruction to the lesser circulation (*e. g.* mitral disease, emphysema, phthisis). Very rarely extreme dilatation of the vessel is followed by semilunar incompetence, when a diastolic murmur at the pulmonary orifice (second left interspace) becomes audible.

Aneurysms involving the pulmonary artery are quite rare: such as occur are small and of the saccular and fusiform varieties.

The **symptoms** resemble those of *intrathoracic aneurysm*, though they are rarely well marked, owing to the fact that they remain of small size as a rule.

**Physical Signs.**—Pulsation (and, rarely, a small tumor) is detectable in the second left interspace. *Palpation* may also render appreciable a thrill and diastolic shock. Coextensive with the area of pulsation there may be *dulness* on percussion, and over the second interspace to the left of the sternum a loud *superficial systolic murmur* is heard on auscultation, together with a diastolic shock. Before attaining to a large size, these aneurysms usually *rupture* into the heart itself.

The **prognosis** is altogether unfavorable, the treatment having reference to the principles that are appropriate in thoracic aneurysm.

The **coronary arteries** may be the seat of aneurysm, though exceptionally. The condition arises in consequence of weak points (due to arteriosclerosis) in the course of the vessels, and is unrecognizable during life.

#### ANEURYSM OF THE CELIAC AXIS.

This condition is sometimes observed in combination with aneurysm of the upper portion of the abdominal aorta.

#### ANEURYSM OF THE SPLENIC ARTERY.

This branch of the celiac axis is occasionally the seat of aneurysmal dilatation. It may be single or multiple, and, whilst it is small as a rule, may in rare cases be quite large.

The **symptoms** are indefinite, but distressing. Deep-seated abdominal *pain*, which shows a tendency to radiation, forms, with *vomiting*, and rarely *hematemesis*, the main features. By careful *percussion* a tumor may be mapped out in the left hypochondriac region, the dulness merging with that of the spleen and the left lobe of the liver. Usually, pulsation, and, rarely, a tumor can be *felt*, and *systolic murmur* is often, though not invariably, heard. The condition must not be confounded with *gastric ulcer*.

#### ANEURYSM OF THE HEPATIC ARTERY.

This is exceedingly rare, the total number of cases on record being about 20. H. B. Schmidt has recently reported a case associated with



symptoms of gall-stones, in which, as shown by the autopsy, death was caused by rupture of the sac into the bile-ducts. Schmidt found records of but 5 cases of this mode of termination. Osler and Ross have reported an instance associated with multiple hepatic abscesses.

The **symptoms** are, in the main, *colicky pains, vomiting, hematemesis, and obstructive jaundice*. A tumor is rarely discernible, though an abnormal area of pulsation is relatively more frequent. The recognition of the condition during life is entirely conjectural.

**Aneurysm of the superior mesenteric artery** is of rare occurrence. *Pain* in the epigastric and lumbar regions, and *demonstrable tumor* near to or directly over the median line of the abdomen, are the symptoms displayed. Detached fragments of the clot may produce *embolism* of the terminal branches of the mesenteric arteries. The condition terminates usually in rupture into the peritoneal cavity.

**Aneurysm of the inferior mesenteric artery** runs a course similar to that of the superior mesenteric, but is so rare as to possess little or no clinical interest.

**Aneurysm of the Renal Arteries.**—Small multiple dilatations are occasionally seen, but large ones are of great rarity. They are prone to rupture into the retroperitoneal cavity.

#### ARTERIO-VEINous ANEURYSM.

**Definition.**—An artificial communication between an artery and a vein. A sac may intervene between the two vessels (*varicose aneurysm*) or there may be a direct fistulous communication without an intervening sac (*aneurysmal varix*).

In varicose aneurysm the sac is developed from the structures that mark the boundaries of the communicating duct. The majority of cases are caused by the simultaneous wounding of an artery and a vein during venesection. Hence their most frequent seat is at the bend of the elbow. Pepper and Griffith have analyzed the records of 29 cases in which the ascending portion of the aortic arch had opened into the vena cava.

**Symptoms.**—The symptoms are to a large extent *aneurysmal*, and in addition there appear in rapid sequence *great swelling of the veins, cyanosis, and edema* of the upper portion of the body. A continuous *thrill* and *buzzing murmur*, with systolic intensification, are the chief physical signs.

In the **treatment** of thoracic arterio-venous aneurysm the same general plan is to be pursued as advised in the purely arterial variety. The management of both forms belongs to the domain of surgery.

#### CONGENITAL ANEURYSM.

This condition arises because of a defective ante-natal development of the elastic coat. It is often multiple, and the tumors are, as a rule, small, in size ranging from that of a pea to a hazelnut. The most common situations for these growths are the coronary and pulmonary arteries. To Eppinger belongs the credit for having pointed out the fact that the aneurysmal walls consist only of the adventitia and intima. The condition may be met with in children, and rarely in adults.

## PART VI.

# DISEASES OF THE DIGESTIVE SYSTEM.

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## I. DISEASES OF THE MOUTH.

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### STOMATITIS.

#### CATARRHAL STOMATITIS.

(*Stomatitis Erythematosa.*)

**Definition.**—A simple, acute inflammation of the buccal mucous membrane. It is more commonly met with in children than in adults.

**Etiology.**—As a *primary* affection its causes are mainly mechanical and chemical irritation, such as the presence in the mouth of hard and sharp bodies, dental caries, acids, hot or cold food, condiments, tobacco, certain drugs (as mercury), eruption of teeth, and bad feeding, particularly in illy-nourished children. It is the result often of a neglect of the mouth-toilet, leading to the decomposition of accumulated bits of food and mucus and to the production of fungous and bacterial irritation. *Secondarily*, catarrhal stomatitis may be associated with certain of the eruptive fevers (scarlet fever, measles, typhoid), also with gastro-enteric derangements, and may follow, by direct inflammatory extension, upon ulcerative tonsillitis, pharyngitis, and the like.

**Symptoms.**—The *local symptoms* of this affection are those usually seen in an inflammation of a mucous membrane—redness, heat, swelling, and dryness, soon followed by increased secretion and soreness. The lips and gums only, or the membrane of the whole mouth, may be inflamed, and the swollen lips, cheeks, and furred tongue may be indented by teeth-marks. Enlarged and reddened papillæ on the tongue and minute vesicles inside the cheeks and lips from projecting mucous follicles are sometimes seen. These, later, may terminate in simple small ulcers. A craving for cold drinks is nearly always noted, as well as distress and even pain on suckling, mastication, or touching with the inspecting finger, and there is a disagreeable taste due to the perverted buccal secretions. *Chemical examination* of the dribbling saliva shows an acid reaction, with the presence, microscopically, of an excess of desquamated pavement epithelium that has undergone partial fatty degeneration. Leukocytes, micrococci, the leptothrix buccalis, and the remains of food may also be seen (Strümpell). Aside from restlessness and the symptoms common to slight febrile disturbances, the *constitutional condition* is rarely disturbed, except when the stomatitis is sec-

ondary either to inflammations lower down in the digestive tract, or to the specific infectious fevers.

The course of the disease is usually acute, and the duration about one week.

The **differential diagnosis** of catarrhal stomatitis is easily made by inspection of the membrane.

The **prognosis**, though favorable, will vary as to time and severity according to the cause.

**Treatment.**—After proper attention to the hygienic surroundings of the patient and the removal of all irritant influences, the treatment is mainly local. The first indications are to cleanse the mouth and allay the pain, and these may be met by the use of cool solutions of boric acid, sodium bicarbonate, or potassium chlorate, 5 and 10 grains (0.324 and 0.648) respectively to the ounce (32.0) of glycerin and rose-water, as mouth-washes, or for swabbing in the case of infants. When iced drinks are ungrateful and the inflammation is more intense and protracted, the use of hot milk and lime-water, mucilaginous decoctions, and sedative and antiseptic sprays of 1 or 2 per cent. solutions of cocain or carbolic acid are often beneficial; or mild astringents may be needed, as  $\frac{1}{4}$  to 1 per cent. solutions of silver nitrate, alum (5 to 10 grains—0.324 to 0.648—to the ounce—32.0—of honey), and glycerite of tannin (2 drams to the ounce—8.0 to 32.0—of water), especially if there is a tendency to chronicity of the trouble, as in toppers and inveterate smokers. Tender and spongy gums may be relieved by the application of equal parts of the tinctures of myrrh and rhatany on a camel's-hair brush (Strümpell). *General symptoms* as they arise must be met by the remedies rationally indicated. Small doses of aconite or potassium citrate for the pyrexia, with a minimum dose of bromid for irritability and sleeplessness, may be all that is required. The internal use of potassium chlorate in children is not to be recommended in this affection, both because of its deleterious action upon the kidneys, and also because it seems to be unnecessary (Forchheimer; Backader). Sometimes an associated gastro-intestinal catarrh needs correction by the use of laxatives. The administration of bland foods and mild ferruginous tonics should be continued throughout convalescence.

#### APHTHOUS STOMATITIS.

(*Follicular Stomatitis; Stomatitis Aphthosa.*)

**Definition.**—A variety of catarrhal stomatitis that is characterized by the eruption of one or more vesicles upon the edges of the tongue, the cheek, or the lips, rapidly passing into small round, or oval discrete spots that are slightly raised and surrounded by yellowish-white bases with narrow red areolæ.

**Etiology.**—Though more common in children between the ages of two and six years, they are by no means rare in adults. Predisposing influences may be found in the seasons (spring and autumn), malnutrition, tuberculosis, dentition, persistent gastro-enteric disorders, anemia, and the acute exanthemata. The *exciting causes* are supposed to be certain deleterious substances, bacterial or toxic, though no special parasite has yet been isolated.



**Symptoms.**—The herpetic vesicles soon rupture, leaving the aphthous ulcers as described above. They are found singly, or at times as many as twenty in number, pin-head to split-pea in size, inside the lips, especially near the frenum, along the tongue-edges, and sometimes inside the cheeks near the edges of the back teeth. They are exquisitely *tender*, so that almost any motion of the affected parts causes sharp burning pain; nourishment is therefore difficult. Patches of *catarrhal stomatitis*, and even of gingivitis, are seen adjacent to the aphthous spots. There is an increased flow of the secretions of the mouth, and the breath is heavy, though not offensive. *General symptoms*, as slight fever, anorexia, and furred tongue, constipation or diarrhea, and irritability, are usually present, with the additional symptoms of any associated disease that may coexist. Gastro-intestinal affections, though often associated with aphthous stomatitis, are most probably due to the common cause, and are not necessarily the cause of the stomatitis in these instances. In some of the specific infectious fevers many aphthæ may appear and tend to run together; these form large irregular ulcers, and give rise to the *confluent form* of stomatitis aphthosa. The special form known as *Bednar's aphthæ*, occurring in young marantic babes, is a rare condition in America. Large white patches are seen on both halves of the posterior part of the hard palate near the alveolar processes, and these may cause large ulcers and involve the bone. Pressure of the tongue upon the thin mucous membrane during nursing, or some other such form of traumatic irritation, appears to act as the cause. Recovery from this type is rare.

The average *duration* of the ordinary discrete aphthous eruption is from four to seven days; in very ill-nourished and poorly cared-for cases the appearance of successive crops of aphthæ will prolong the distress.

**Diagnosis.**—This is based upon the characteristic appearance of the ulcers and the degree of soreness. Aphthæ must be differentiated sometimes from *thrush*, and the distinguishing features will be dwelt upon in the description of the latter affection. Herpes of the mouth, so called, and aphthous vesicles are probably identical in most cases; and the presence on the lips of herpetic vesicles in some of the grave constitutional diseases indicates simply the severity of the common cause of the aphthæ.

**Prognosis.**—The discrete form is mild, and favorable in its course toward recovery; confluent aphthæ is more troublesome, and follows a prolonged course on account of the general debility induced by the associated disease (Starr). In certain adults, as well as in children, the affection is apt to recur; relapses are also frequent in those having weak digestive and imperfect assimilative functions.

**Treatment.**—It is first necessary to remove all irritating influences, and in order to minimize the intense pain of the aphthous spots the blandest liquids and the softest foods that are consistent with the sustenance of the patient are imperative. Absolute cleanliness of both the foods and the vehicles of administration, especially in bottle-fed children, is not less important than the thorough cleansing of the mouth, particularly after taking nourishment. Local applications are of obvious value. Demulcents, as mucilage of sumac, or of marshmallow, with boric acid (gr. v to ʒj—0.324 to 32.0), sodium bicarbonate (gr. v-x to ʒj—0.324–0.648 to 32.0), carbolic acid, or potassium permanganate (gr. iv

to  $\mathfrak{z}\mathfrak{j}$ —0.259 to 32.0), are invariably useful. Swab-applications of wine of opium ( $\mathfrak{M}\mathfrak{v}$  to  $\mathfrak{z}\mathfrak{j}$ —0.333 to 32.0) or of cocain (4 per cent. solution) may be necessary when the pain is intense, and prior to taking food.

To promote the healing of the ulcers a very light touch with the silver-nitrate stick or solution (gr. x-xxx to  $\mathfrak{z}\mathfrak{j}$ —0.648—1.94 to 32.0) is often beneficial. Much favor is deservedly given also to potassium chlorate in solution (gr. x to  $\mathfrak{z}\mathfrak{j}$ —0.648 to 32.0), or applied in the dry powdered form. In the confluent aphthous ulcer the use of sodium salicylate ( $\mathfrak{z}\mathfrak{j}$  to  $\mathfrak{z}\mathfrak{j}$ —4.0 to 32.0) has been recommended, while an ethereal solution of iodoform ( $\mathfrak{z}\mathfrak{i}\mathfrak{j}$  to  $\mathfrak{z}\mathfrak{j}$ —8.0 to 32.0) has been advised by J. Lewis Smith. For bleeding and spongy gums the mild astringents mentioned in the treatment of catarrhal stomatitis are indicated. Stronger astringents may answer for application to sluggish ulcers; thus copper sulphate, either solid or in solution (gr. x to  $\mathfrak{z}\mathfrak{j}$ —0.648 to 32.0), and zinc sulphate (gr. xv to  $\mathfrak{z}\mathfrak{j}$ —0.972 to 32.0) are useful. Potassium chlorate acts as a specific in ulcers of the mouth, and is eliminated by the buccal secretions, which keep the ulcerated surfaces constantly bathed with the drug, so that its internal use is to be recommended, though in very small doses in children, being given well diluted, as in the following formula:

R $\mathfrak{y}$ . Potassii chlorat.,	gr. xxiv (1.55);
Tinct. myrrhæ,	gtt. x (0.666);
Syr. acaciæ,	f $\mathfrak{z}\mathfrak{i}\mathfrak{j}$ (64.0);
Aquæ dest.,	q. s. ad f $\mathfrak{z}\mathfrak{i}\mathfrak{i}\mathfrak{j}$ (96.0).—M.

Sig. Teaspoonful every three hours for a child three years of age.

Constitutional symptoms are to be combated on general principles and require careful attention. Remedies directed to the correction of digestive derangements and to the stimulation of assimilation are also often required. Good food and ferruginous tonics are frequently necessary.

#### MEMBRANOUS STOMATITIS.

(*Stomatitis Crouposa*.)

**Definition.**—In this form of stomatitis the inflammation is more intense and more extended in area than in the aphthous form, being also attended with the formation of a false membrane, which, when separated from the subjacent mucous surface, leaves a deeper and larger ulcer than does aphthous stomatitis.

The **pathology** of these membranous patches, which are very seldom confined to the mouth alone, is embraced in the article on Diphtheria. If in the latter disease the typical false (diphtheritic) membrane is removed, it leaves a deeper ulcer than does the removal of a croupous membrane, in which the coagulation-necrosis involves the more superficial layers. In cases in which necrosis of the upper layers of the oral mucosa follows from the application of caustics, the coagulation-process extends inward from the surface, forming yellowish-white patches of dead tissue, which, on separation, leave an ulcer (Allichin). Membranous stomatitis may also be due to gonorrheal or syphilitic infection of the new-born.

The **etiology** of membranous stomatitis is, then, either specific (diphtheritic,) or non-specific, due to chemical or physical irritants.

The account of the local symptoms and treatment is also included in the description of the causal diseases.

#### ULCERATIVE OR FETID STOMATITIS.

(*Stomatitis Ulcerosa*.)

**Definition.**—A specific ulcerative inflammation of the buccal mucous membrane and gums, attended with marked fetor of the breath, and having a tendency to extend widely and deeply.

**Etiology.**—The predisposing causes of this malady are principally as follows: Childhood, after the commencement of the first dentition, and usually between the ages of three and eight years; damp weather, especially during spring and autumn; unhygienic surroundings, particularly the lack of pure air, of good and abundant food and clothing, and the added detriments to health for which neglect and filth, specific infectious diseases, uncleanness of the mouth, caries and loosening of the teeth, and congenital heart-disease (Duckworth) are responsible. An endemic type of this affection has been observed among soldiers in camps and barracks, among children in crowded eleemosynary institutions, and in jails, and may be accounted for by unsanitary conditions affecting communities alike; its epidemic and contagious character likewise points to a microbic origin. The specific *exciting cause*, it has been held, corresponds to the hoof-and-mouth disease of cattle, the poison being conveyed in milk. Payne suggests the identity of the virus with that of *impetigo contagiosa*. The careless administration of mercury, especially to susceptible adults, may also be followed by a severe mercurial stomatitis that is typically ulcerative. Scurvy (*scorbutic stomatitis*) and the persistent use of lead and phosphorus are also excitants of this disease.

**Clinical Symptoms.**—*Locally*, the disease starts, as a rule, at the edges of the gums opposite the lower incisor teeth, gradually spreading backward and to the adjoining portions of the lips and cheeks. The gingival mucous membrane is deeply red and swollen; the gums soon become spongy, bleed easily, and break down into thick, soft, grayish sloughs, which leave deep and ragged ulcers surrounding the necks of the teeth. The latter even become loosened, and in protracted cases the alveolar periosteum may become inflamed and cause necrosis of the bone. Profuse salivation, a foul breath (that once earned for the condition the term of “putrid sore mouth”), occasional slight hemorrhages from the gums, and excessive discomfort, or even pain, on mastication are nearly always present. The tongue is coated, swollen, and tooth-marked; aphthæ are sometimes seen, and the submaxillary glands are generally swollen. The *general symptoms* attending this ailment are those of a lowered state of vitality, produced by an unhygienic environment, or cachexia, or severe illness primary to it, with, usually, moderate fever. Nausea and vomiting or an offensive diarrhea may supervene as the result of swallowing the putrid discharges.

**Course and Duration.**—Though acute in its course, the highly debilitating character of the disease may tend to make it chronic, especially when there is alveolar necrosis and a neglect of proper treatment. Ordinarily, with careful management, convalescence may be



established in from four days to a week. Goodhart regards the occasional termination of the pyrexia by lysis, with an accompanying improvement of the local symptoms in such cases at least, as suggestive of the specific nature of ulcerative stomatitis.

**Diagnosis.**—Upon examining the mouth and noting the characteristic ulceration, the fetid breath and saliva, and the cachectic appearance, the disease is usually recognized, and should not be confounded with the dark, extensive, gangrenous sloughs of noma.

The **prognosis** is favorable in typical cases, though less so in marasmic and neglected cases and when cancrum oris or necrosis of the jaw are superadded; in such cases recurrence, chronicity, deformity, and even death, may take place.

**Treatment.**—It is well in nearly all ill-nourished, uncleanly-kept, and sickly children, as well as in cases in which mercury is to be administered for any length of time, to prescribe mouth-washes of potassium chlorate (gr. xv to ʒj—0.972–32.0), in order to prevent the occurrence of mercurial or ulcerative stomatitis. The hygienic treatment of ulcerative stomatitis is important. On account of the contagiousness of the disease cases occurring in a family or in institutions should be isolated, and fresh air, light nourishment, and cleanliness are *sine quâ non* of recovery. The local treatment is virtually a specific one in the use of potassium-chlorate washes (gr. x–xx to ʒj—0.648–1.296 to 32.0—of rose-water or demulcent), aided by the internal administration of the same salt in small doses. For the disagreeable fetid odor the alternate use of antiseptic washes is indicated. Solutions of carbolic acid or potassium permanganate, in strength equal to or slightly over that indicated in the treatment of aphthæ, or hydrogen peroxid (ʒj–iij to ʒj; 4.0–12.0 to 32.0), or listerin and water (equal parts), are useful. Pencilling the spongy gums with such astringents as tincture of rhatany, silver nitrate (gr. x to ʒj—0.648–32.0), alum, and also with tannic-acid solutions, may be necessary. Loosened teeth should not be disturbed, as they may grow firm with convalescence, though surgical interference may be required in cases of necrosis of the alveolar process. Until the patient has regained considerable vigor the use of mild antiseptic mouth-washes should be continued regularly, so as to effect thorough cleanliness and rid the oral cavity of lurking germs. Careful attention to the teeth is always requisite. During the height of the disease constitutional treatment may have to be directed toward stimulating the languid and lowered vitality. For this purpose either whiskey or brandy, in half or one teaspoonful doses in milk, is extremely useful; the elixir of cinchona, with some vegetable salt of iron, as the citrate or tartrate, also forms a useful combination. When there is pyrexia or a diminished urinary secretion the internal use of potassium chlorate is dangerous and must be cautiously employed. The following prescription is tonic and almost specific, and may be recommended:

R̄. Potassii chloratis,	gr. xlviij (3.11);
Acidi hydrochlor. dil.,	fʒj (4.0);
Syrupi,	fʒvj (24.0);
Aquæ destillat.,	q. s. ad fʒiij (96.0).—M.

Sig. Teaspoonful diluted, every two hours for a child three years old (Starr).

The prolonged use of tonics and cod-liver-oil emulsion with lime-salts in scrofulous, rachitic, and scorbutic subjects must be carried on in order to prevent relapses of ulcerative stomatitis.

#### PARASITIC STOMATITIS.

(*Thrush ; Stomatitis Mycosa.*)

**Definition.**—A specific, contagious fungous disease, characterized by the rapid formation upon the oral mucous surfaces of small, whitish, soft, and lightly adherent spots or flakes, tending to coalesce and spread throughout the entire buccal cavity.

**Etiology.**—Predisposing causes are—infancy with its concomitant disorders of the gastro-intestinal tract (especially when unhygienic surroundings and a consequently impaired vitality and malnutrition prevail), also congenital syphilis, tuberculosis, and the exanthemata. The disease may attack adults and complicate the typhoid and cachectic states, as in the final stages of low fevers, carcinoma, chronic tuberculosis, and diabetes. The growth of thrush-patches is due, specifically, to the *saccharomyces albicans* (formerly *oidium albicans*). It is a characteristic of this fungus to develop from round or oval spores in the formation of long-branching mycelium filaments, from the ends of which a multiplication of ovoid torulae-cells takes place by the process of simple budding. These mycelia exhibit a tendency to penetrate the deeper layers of the mucosa of the mouth and also into the mucous glands (Starr). Since the growth of this organism requires both an altered condition of the mucous membrane and an acid medium, the primary or exciting cause of thrush is to be found in whatever produces such a favorable nidus. Most important in this connection is uncleanness, particularly in the case of poorly-nourished and bottle-fed children. The development of catarrhal stomatitis and the acid fermentation of remnants of food (especially of saccharine substances), which prevent the nutrition of the mucosa and acidify the normally alkaline oral secretions, are common causes of thrush. The further growth of the fungous patches also contributes to the acid state of the already abnormal buccal fluids. The fact that the spores of thrush may be transferred to other cases by bottle-tips, spoons, and ill-kept feeding-bottles is well recognized as an explanation for the occasional endemic character of the malady.

**Symptoms.**—Any marked local symptoms are due rather to the coexisting stomatitis than to the thrush itself (Allchin). There will be some *soreness, heat, persistent dryness, and lividity* of the mucous membrane. Thrush-spots, slightly raised above the surface, begin to appear on the tongue, and grow into patches that may coalesce and spread to the cheeks, lips, and hard palate: they may even invade the tonsils, pharynx, and esophagus, and, rarely, the true vocal cords, the stomach, and cecum (Parrot). At first pearly-white in color, the curd-like flakes may become yellow and even brown, owing to slight hemorrhages caused by the irritation. Though early adherent, in a few days they become loose, and when brushed off leave a smooth surface; when complicating some serious gastro-intestinal disease or dyscrasia, however, their attachment is deeper, and the deposit may sometimes appear in successive crops. A *microscopic examination* of the thrush-patches shows inter-

lacing, irregular, and branched mycelial threads, spores, occasional bacilli, and leptothrix-filaments imbedded in a mass of granular débris and fetid particles. The buccal fluids are acid in reaction. The *general symptoms* depend upon the associated disease, and are usually those of wasting, artificially-nourished children having digestive troubles or a constitutional taint.

**Diagnosis.**—This may be accurately made upon the discovery of the fungus by microscopic examination. Only very rarely are portions of the thrush-organism found in the false membrane of *diphtheritic stomatitis*. *Milk curds* may be readily removed, and are not necessarily associated with the stomatitis accompanying thrush or the grave systemic states. The only important point of differential diagnosis arises in the case of *aphthæ*. The following table will express the main points upon which a safe discrimination may be based :

PARASITIC STOMATITIS (THRUSH).	APHTHOUS STOMATITIS.
Dryness of the mouth.	Salivation.
Whitish, raised spots or patches with no red areola ; these are easily removed, leaving no ulcer and causing no bleeding.	An ulcer with a yellowish-white, depressed base, surrounded by a red areola. The base is removed with difficulty by forceps, and bleeding results.
Spots are numerous.	Usually few in number and discrete.
Begins in the form of minute spots.	Not so ; ulcers appear, preceded by the formation of herpetic vesicles.
Ulcers not painful. Discomfort depends on the associated stomatitis.	Ulcers exquisitely tender.
The characteristic thrush-fungus is always present in the deposit, and can be seen with the microscope.	No specific micro-organism determined, though probably present.

**Prognosis.**—This is favorable as regards the thrush alone, but, occurring in marantic children and cachectic adults, its appearance is of grave significance, and it is even suggested by some that it portends a speedy death.

**Treatment.**—Prophylaxis is of great moment, since it is much easier to keep the mouth clean and the secretions normal, and to attend to proper food, and thus avoid creating a soil for the growth of the vegetable parasite, than it is to prevent absolutely the entrance of thrush-spores. Efforts directed toward preventing acidity are especially indicated. This is to be done by the use of mild alkaline mouth-washes, as soda-water and lime-water. The dietary should be carefully looked after, and should exclude sugars and all starchy food ; the addition of lime-water to the milk (about one part to four) is a desirable precaution to take, particularly with children. Cleansing the feeding-apparatus and the mouth after each feeding is essential, both in the prevention of the formation, and in decreasing the further growth, of thrush when present. The local treatment consists in the use of alkaline and antiseptic applications, preferably by means of the spray. Solutions of boric acid or sodium hyposulphite ( $\bar{5}j$ —4.0—of either to  $\bar{5}j$ —32.0—of water, with the addition of a little glycerin), potassium permanganate, or hydrogen peroxid, are useful. Syrupy excipients are to be excluded. Potassium chlorate may exert a beneficial effect in those cases in which stomatitis is associated, as may also pencilling with a solution of silver nitrate (gr. v to  $\bar{5}j$ —0.324 to 32.0) over the inflamed mucosa.



When esophageal obstruction exists it may be necessary to gently force a rubber tube through the mass of thrush-deposit in order to give nourishment (Forchheimer).

**Medicinal treatment** embraces the administration of nourishing and easily digestible food, occasional stimulation, and the correction of gastro-intestinal disorders. Attention must also be paid to the primary affections to which the thrush is superadded. Iron, cod-liver oil, and acid and bitter tonics in palatable form are usually indicated in debilitated subjects, along with general hygienic measures. The internal use of small doses, frequently repeated, of calomel or mercuric chlorid may also be tried for a possible specific effect in combating thrush.

#### LA PERLÈCHE.

This contagious disease is confined to the angles of the mouth. It was first described in 1886 as prevalent among the children of Limousin in France by Lemaistre. It was found that the drinking-water in that locality contained cocci similar to the spherobacteria that infested the epithelial thickenings, and that these were probably conveyed to human beings by drinking-vessels. Little elevations and fissures, said to resemble those of congenital syphilis, were seen around the oral angles. The latter were the seat of smarting pain, particularly on opening the mouth suddenly or too far, and caused the patient to lick (*perlîcher*) them constantly. The disease seemed to be entirely local, and lasted from two to three weeks. Alum and copper-sulphate solutions were most useful.

#### GANGRENOUS STOMATITIS.

(*Noma; Cancerum Oris.*)

**Definition.**—A rapidly-spreading gangrenous affection of the cheek and gums, of rare occurrence, usually asymmetric, and ending fatally in most cases.

**Pathology.**—In addition to the necrotic changes in the cheeks, the process may extend to the jaws and lips. The blood-vessels contain thrombi, thus preventing hemorrhage from the sphacelus. The submaxillary and cervical glands may be slightly enlarged and soft. Blood-changes of an uncertain character have been noted. Hemorrhagic infarctions, aspiration broncho-pneumonia, or gangrene by inhalation of gangrenous particles or metastasis, may be met in the lungs. Wharton has described an associated membranous form of colitis, and a metastatic infiltration of the cardiac muscle and purulent pericarditis may also be seen *post-mortem*. Klementorsky met with a peculiar and fatal form of gangrene limited to the gums of babes and occurring a few days after birth.

**Etiology.**—*Predisposing Causes.*—This uncommon affection attacks girls more frequently than boys, usually between the ages of two and five years; it appears to be endemic in low, moist countries, as Holland, though apparently it is not contagious. Sickly and ill-nourished children suffering from the effects of overcrowding and previous disease are especially liable to noma. Most often, however, it is secondary to measles; it may also follow scarlet fever, typhoid, small-pox, or less frequently pertussis. Boydon reported a case associated with erysipelas

in an adult. The causative influence of mercurialization and ulcerative stomatitis has been overrated. The primary *exciting cause* of cancrum oris is probably microbic, and Lingard has found "long thread-like growths made up of small bacilli at the line of extension of the necrotic patch."

**Symptoms.**—The mucous membrane of one cheek, near the corner of the mouth, is usually first affected, a *dark, ragged, sloughing ulcer* appearing and spreading insidiously for two or three days before the substance of the cheek is involved. A *hard and sensitive nodule* may then be felt by grasping the cheek between the thumb and finger on placing the one within the mouth over the ulcer and the other outside. Brawny induration of the skin over this nodule soon becomes manifest, and then there appear collateral edema and an unctuous-looking, deeply livid, gangrenous spot, soon becoming bullous and leaving a black eschar. Perforation of the cheek may occur on the third day, though usually not until a week has passed. There is an *ichorous discharge* of shreds of gangrenous tissue from the unhealthy wound. The *fetor* of the breath is almost intolerable and characteristically gangrenous. The necrosis may extend over one-half the face of the side affected, and may involve the gums and jaws, but seldom does it attack the opposite side of the face. The *general symptoms* of such a grave malady may be slight at a very early period, but with the formation of the eschar they become rapidly severe and typhoid in type. Great prostration, delirium, pyrexia (104° F.—40° C.), diarrhea, and edema of the feet are common. The course is actively acute; the duration rarely extends beyond two weeks.

**Complications.**—Septic lobular pneumonia may occur from aspiration of gangrenous particles; colitis and gangrene of the genitalia in females (*noma pudendæ*) are also seen. In those very rare cases that recover granulations form, the gangrenous edges become clean, and cicatrization, with great disfigurement of the face and even restricted jaw-motion, is then apt to follow.

**Diagnosis.**—The disease when fully established is easily diagnosed by its characteristic origin, the gangrenous ulcer-nodule, the eschar-formation, and perforation, associated with a previous history of measles or other acute infectious fever of childhood. The offensive fetid odor and severe constitutional depression are also of great value.

**Differential Diagnosis.**—From *anthrax* it differs in that the latter affection is more common in adults, with a history of contagion, and in the fact that malignant pustule starts on the exterior of the cheek, and perhaps in a previous abrasion in the skin. The discovery of the bacillus anthracis in the blood and discharges is conclusive. *Ulcerative stomatitis* of a severe and neglected type may be confounded with cancrum oris, but in the former the destruction of tissue is mainly of the gums and alveoli, the cheeks being simply ulcerated and no extensive sloughing taking place; the breath, though fetid, is not gangrenous, and the oral discharge, though sometimes bloody, is not mixed with shreds of gangrenous tissue (Starr). Finally, the course of ulcerative stomatitis is less severe, a fatal termination being extremely rare.

**Prognosis.**—Noma is seldom recovered from, the mortality being about 80 to 90 per cent. (Bogel). When recovery does take place the

development of ectropion, facial deformity, and local disability, with a protracted convalescence, render life burdensome.

**Treatment.**—This embraces the prevention of gangrenous stomatitis by means of a proper management of the diseases that are known to cause it; careful hygiene and the avoidance of mercurialization will also be of undoubted use. The primary indication in the *local treatment* is the arrest of the gangrenous process, thus causing, if possible, a healthy reaction on the part of the surrounding tissues. All dead sloughs should be cut away before using escharotics, and with this end in view some recommend the prompt application of strong caustics, as fuming nitric acid, the acid nitrate of mercury, solid zinc chlorid, silver nitrate, carbolic acid, a concentrated solution of perchlorid of iron, Vienna paste, and the actual cautery. For the protection of the healthy parts and for efficiency the Paquelin or the galvanic cautery is probably best. Anesthesia is requisite for such strong measures. Milder applications, however, seem to be quite adequate in some cases. Thus, bismuth subnitrate, potassium chlorate, and aristol, or the following formula by Dr. Coates, may be tried:

R̄. Cupri sulph.,                      ʒij (8.0);  
     Pulv. cinchonæ,                    ʒss (16.0);  
     Aquæ,                              q. s. ad fʒiv (128.0).—M.

It is essential, for the prevention of septic infection to ensure cleanliness of the wound and of the mouth, and to promote the separation of the sloughs. To effect the former we employ mild antiseptic washes of carbolic acid, hydrogen peroxid, Labarraque's solution, potassium permanganate, etc.; for the latter and for the diminution of the fetor, antiseptic charcoal poultices containing boric or salicylic acid are useful. Mild antiseptic and astringent lotions of boric acid, zinc sulphate (gr. ij to ʒj—0.129 to 32.0), or balsamic ointments with vaselin, may aid in healing the granulating surfaces in favorable cases. The internal treatment must be directed toward sustaining the strength of the patient by the administration of the most nourishing food, stimulants, and tonics, especially quinin, iron, and strychnin. Rectal feeding may be necessary. Plastic operations are sometimes needful after recovery to mitigate oral disabilities or facial deformities resulting from cicatricial adhesions and contractions.

#### MERCURIAL STOMATITIS.

(*Mercurial Ptyalism.*)

**Definition.**—An inflammation of the mouth and salivary glands, caused by the excessive use of mercury; it is rarely seen as a result of the therapeutic use of other drugs.

**Etiology.**—Predisposing causes are dyscrasia and occupation, mainly. The peculiar individual susceptibility of these subjects to dyscrasia will not permit the use of even minimum doses of mercury without serious and almost immediate symptoms of ptyalism. This is also seen in barometer-makers, mirror-silverers, chemists, and others who handle mercury in their daily work. The exciting cause of ptyalism is the ingestion, inhalation, or cutaneous absorption of mercury.



**Symptoms.**—A *metallic taste* in the mouth is first noticed by the patient. Soon the *gums* become “touched”—*i. e.* red, swollen, tender to the touch, and sore during the act of mastication. A marked secretion and *flow of saliva*, with a *fetid breath* and swollen tongue, follow. Very rarely in this disease the affection passes into an *ulcerative stomatitis*, and causes loosening of the teeth and necrosis of the maxilla. *General symptoms*, as constitutional depression, anorexia, diarrhea, mental anxiety, and nervousness, may supervene.

The recognition of the foregoing causal factors—predisposing and exciting—renders the *diagnosis* easy. The *prognosis* is favorable, and, although the local symptoms may be harassing, recovery is attainable within a few weeks as a rule.

**Treatment.**—The toxic action of mercury in the production of pytalism can be avoided by a knowledge of individual susceptibility and by the local and internal use of potassium chlorate. Upon the first appearance of the symptoms there must be a prompt withdrawal of the mercurial influence, and a change of occupation if that be the predisposing cause. Locally, soothing, alkaline, and mildly antiseptic mouth-washes, as in the treatment of catarrhal stomatitis, may be all that is necessary. For the fetid breath solutions of boric acid or potassium chlorate may be used. Ulcers may be brushed with silver-nitrate solution. The internal treatment should be directed toward keeping the bowels soluble; in addition, alkaline mineral waters may be used, and in severe cases potassium chlorate in 5- to 10-grain (0.324–0.648) doses. Atropin (gr.  $\frac{1}{100}$ —0.0006) and opium have been recommended to decrease the excessive salivary secretion and to allay pain, and hot baths will aid the treatment materially. In severe cases the resulting debility and anemia should be met by the use of highly nourishing liquid foods and by tonics.

Osler points out that the condition of the teeth known as *erosion*, which sometimes follows infantile stomatitis, and especially the mercurial form, is to be discriminated from the deformed teeth of congenital syphilis. In the former the first permanent molars, and then the incisors, are observed to have small pits or discolored and eroded spots, due to a morbid deficiency in enamel-formation. The notched and irregular teeth of hereditary syphilis in children (Hutchinson) are sufficiently distinctive.

## II. DISEASES OF THE TONGUE.

### GLOSSITIS.

#### ACUTE GLOSSITIS.

(*Glossitis Acuta.*)

**Definition.**—An acute parenchymatous inflammation of the tongue, sometimes ending in abscess.

**Etiology.**—Predisposing causes are supposed to be an impaired general health and exposure to cold, humid weather. The exciting causes

are most frequently the stings and bites of insects, or burns, scalds, and the action of corrosives. I believe that many cases follow slight injuries to the tongue that allow of the introduction of inflammatory poisons or microbes.

**Symptoms.**—These come on *rapidly* and with more or less local severity and danger. The *tongue* becomes much swollen, and may even protrude beyond the lips. It is very *tender* and *painful*, and coated with a thick, soft yellowish-white fur, and it may also be dry, cracked, and ulcerated. *Catarrhal stomatitis* is often associated, salivation is usually profuse, and talking, swallowing, and even breathing are rendered difficult and distressing. *Dyspnea*, even to suffocation, may be imminent. The cervical and sublingual glands may be swollen, moderate *fever* is always present, and the obstruction to breathing and administration of nutriment may assume a dangerous aspect.

The inflammation reaches its height in about three or four days, tending to subside almost entirely about the seventh day. Not rarely the inflammatory infiltration passes into suppuration with the formation of a circumscribed abscess of variable size in one-half of the tongue; fluctuation may not, however, be obtainable, spontaneous rupture being sometimes the first indication of abscess. The **prognosis** is favorable, except that serious obstruction is likely to remain.

**Treatment.**—When the case is seen quite early and during the congestive stage, the topical use of ice, allowed to slowly dissolve in the mouth, may be both grateful and of service in preventing the swelling. Mucilaginous mouth-washes, containing some mild antiseptic, as sodium borate with sodium bicarbonate (gr. v–xx to  $\bar{3}j$ —0.324–1.296 to 32.0), should also be employed. A brisk saline purge, given early, will aid in reducing the inflammation, and should the tongue become alarmingly swollen, deep scarification and the use of half a dozen leeches between the hyoid bone and the jaw-angles may be of decided service. Steam-atomization, medicated with the compound tincture of benzoin or ammonium chlorid ( $\bar{5}j$  to  $\bar{3}j$ —4.0 to 32.0), favors resolution (Cohen). Abscesses must be incised and washed out with antiseptic solutions. Tracheotomy is rarely called for to relieve the dyspnea. Rectal alimentation with predigested foods may be necessary, and during convalescence ferruginous tonics in glycerin and bland foods should be continued for some time, in order to prevent chronic inflammation and thickening. Any local source of irritation, as from carious or sharp teeth, should be removed.

#### CHRONIC SUPERFICIAL GLOSSITIS.

**Definition.**—A chronic inflammation of the mucosa of the tongue.

**Etiology.**—This disease is often preceded by several acute attacks, the habitual use of tobacco, both in smoking and chewing, and of strong spirituous liquors being mainly productive of the original affection. The frequent use of irritating foods is also a prominent factor in some instances.

**Symptoms.**—The surface of the tongue is continually sensitive and more or less reddened. Often there are seen ovoid patches of various size, smooth and shiny, on account of the loss of papillæ, and separated

by furrows that extend to the depth of the mucosa itself. The tongue may also be slightly furrowed in intervening spaces, especially at the base. The general health is somewhat deteriorated.

**Diagnosis.**—This rests upon the history of the case and upon the results of examination of the organ.

The **prognosis** is favorable as to alleviation, but guarded as to cure.

**Treatment.**—The blandest dietary must be insisted on, as well as absolute abstinence from the causal irritants, exacerbations being prone to occur. The local use of demulcents and of mildly alkaline and antiseptic lotions, such as Seiler's tablets in solution, and of solutions of chromic acid or silver nitrate (gr. v-x to ʒj—0.324-0.648 to 32.0) in glycerin or honey, applied once or twice daily by gentle brushing, is to be recommended. General tonics and the avoidance of irritating drinks will be indicated.

#### GLOSSITIS DESICCANS.

A rare disease, chronic in nature and of unknown causation. It is characterized by "the gradual development upon the surface of the tongue of a number of deep fissures and indentations, giving the organ an uneven and ragged look. The pain is due to the frequent presence of excoriations and ulcers in these fissures" (Strümpell). The *prognosis* of the affection is favorable as regards any danger. The *treatment* is hygienic, consisting of cleanliness of the mouth and the use of disinfectant mouth-lotions, together with the topical use of alterative or astringent applications, as silver nitrate or chromic acid, to any ulceration.

#### LINGUAL PSORIASIS (TYLOSIS LINGUÆ).

In this disease there are small regular areas of hyperplasia of the glossal epithelium, eventually causing a map-like appearance of the surface of the tongue—"lingua geographica." The trouble is obscure in its etiology and persists for years. Seldom is there any discomfort associated, although mental anxiety or hypochondriasis may develop.

#### LEUKOPLAKIA ORIS (BUCCAL PSORIASIS).

In this affection the mucous membrane of the mouth and tongue may be involved. On the lateral borders of the tongue white or bluish-white scar-like spots or patches, often slightly notched, make their appearance. Some of these pass away to be replaced by others, and the affection progresses despite all attempts to cure it. The true cause is unknown, but it has been suggested that some irritant, as the use of a pipe, may account for the condition. The malady has, however, been seen in women. A syphilitic taint is said to especially predispose to the disease (Strümpell). The affection must be carefully diagnosed from the oral manifestations of syphilis, if for no other reason than to relieve the mind of a morbidly anxious patient. Excepting some pain connected with possible ulceration, there are no annoying symptoms, and the treatment further than that suggested for glossitis desiccans is usually of no value.



In children a similar tongue-affection has been named "wandering rash." The whitish patches are circinate and enlarge peripherally, forming rings of epithelial hyperplasia, within which is a red, glossy center "devoid of filiform papillæ, though the fungiform remain" (Allchin). The affection is regarded as a tropho-neurosis.

#### ANGINA LUDOVICI.

(*Ludwig's Angina.*)

**Definition.**—A rare acute phlegmonous inflammation of the floor of the mouth.

**Etiology.**—The condition may be idiopathic, but more often it is secondary to specific infectious diseases, as scarlet fever and diphtheria. Undoubtedly it is directly caused by a streptococcus and by an extension of the infection from adjacent glands (Osler).

**Symptoms.**—These are *intense* at the *outset*, and begin with swelling in the region of the submaxillary gland, with a rapid involvement of the cellular tissue of the floor of the mouth as well as of the anterior portion of the neck. *Pain* is marked, and this, with the *acute swelling*, renders articulation, mastication, and deglutition extremely difficult. Compression or edema of the larynx may often cause dangerous dyspnea. The *constitutional disturbance* is usually febrile, and may either approach the typhoid type or may be septic. The condition generally terminates either in abscess or extensive sloughing (*cynanche gangrenosa*), and only rarely does resolution take place.

The **diagnosis** is easily made when complicating a specific fever.

The **prognosis** is always guarded, since death sometimes occurs. Relapses are likewise apt to follow in weakly and strumous subjects.

**Treatment.**—The most that can be done is to sustain the strength of the patient and secure prompt surgical interference when the process has reached the point of beginning suppuration or gangrene. Tracheotomy may be demanded if asphyxia threatens life.

### III. DISEASES OF THE SALIVARY GLANDS.

#### HYPERSECRETION.

(*Ptyalism.*)

**Definition.**—An abnormal increase in the secretion of saliva.

**Etiology.**—Salivation as an idiopathic affection is rare, and as such is considered to be a neurosis. Thus, it has been seen in emotional children of from two to eight years of age, though apparently in perfect health. According to Bohn, the secretion in these cases is mostly increased during active exercise, is reduced on lying down, and absent during sleep. Spontaneous recovery takes place in a few years. As a deuteropathic disease ptyalism may be the result of oral disease (*e. g.* noma, ulcerative stomatitis), and also of gastro-enteric, pancreatic,

uterine (as gestation), centric (as diseases or tumors of the medulla or of the facial nerve), toxic, systemic (as small-pox, the use of mercury, iodids, pilocarpin, tobacco), and hydrophobic irritation and disease.

**Diagnosis.**—It should be pointed out that a failure in swallowing the normal quantity of saliva may cause dribbling from the mouth and simulate true hypersecretion.

The **prognosis** is favorable in itself, but dependent on the gravity of the cause.

**Treatment.**—The causes are to be removed and the general health toned up. For stomatitic salivation potassium chlorate is first in rank as an internal and local remedy. Iron and arsenic are valuable in neurotic cases, and the bromids or hyoscin may be of supplemental use. Atropin (gr.  $\frac{1}{200}$  to  $\frac{1}{100}$ —0.0003 to 0.0006) and belladonna are almost uniformly successful in idiopathic as well as in central ptyalism.

## XEROSTOMA.

(*Aptyalism*; “*Dry Mouth.*”)

**Definition.**—A morbid suppression or arrest of the salivary and buccal secretions.

**Etiology.**—Most of the cases of dryness of the mouth have been observed in women in conjunction with nervous or sudden mental phenomena, and only as a temporary condition. Centric involvement connected with the secretion of saliva is probable in some cases (Hadden). Much more commonly xerostoma is an effect of the febrile state, of mouth-breathing (due to nasal obstruction), and of diabetes.

**Symptoms.**—Apart from the sensation of dryness, mastication, deglutition, and articulation are difficult. The *local appearances* show a glazed, shiny, red, and sometimes cracked condition of the tongue and labial and palatine mucous membrane. The absence of saliva may permit the remnants of food to collect around the gums and harden (Osler).

The **diagnosis** is made on inspection, the **prognosis** depending on the removability of the cause, and rightfully being guarded on account of the frequent obstinacy of the trouble.

**Treatment.**—Attention to the systemic condition, whether diseased or merely debilitated, is requisite. Small doses of potassium iodid and pilocarpin (gr.  $\frac{1}{20}$ —0.003) in gelatin lamellæ or in lozenge form, allowed to dissolve in the mouth with the aid of a sip of water, have been productive of relief. In cases of obscure or of centric origin the galvanic current should be tried.

## SYMPTOMATIC PAROTITIS.

(*Parotid Bubo.*)

**Definition.**—A secondary inflammation of the parotid gland, generally due to septic infection, usually unilateral, and tending to suppuration.

**Etiology.**—Not being a primary affection, the causes giving rise to

it may be mentioned as follows: (a) Acute infectious fevers, as typhoid, typhus, pneumonitis, pyemia, erysipelas; (b) Injury or disease of the abdomen or pelvis (Stephen Paget), especially when associated with the genito-urinary tract, as mild traumatism or derangement of the testes or ovaries, the use of a pessary, or even menstruation or pregnancy; gastric ulcer may be accompanied by it; (c) Peripheral neuritis with facial paralysis (Gowers).

Most of the cases are probably septic and indicative of an unfavorable course in the progress of the associated disease, and especially of the fevers mentioned. The *symptoms, diagnosis, and treatment* of the parotitis itself fall more properly under the scope of surgery.

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## IV. DISEASES OF THE TONSILS.

### ACUTE TONSILLITIS.

**Definition.**—An acute inflammation of the tonsil or tonsils, affecting either the mucous membrane, the follicles, or the parenchyma, and ending either in resolution, suppuration, or chronic enlargement.

**Pathology.**—In the *superficial variety* of acute tonsillitis the mucosa is simply red, swollen, and sometimes covered with a thin, soft exudate of muco-pus. The tonsil itself may also be swollen. In *follicular tonsillitis* the lacunæ become filled with a cheesy exudate which often protrudes from the tonsillar crypts; epithelial and pus-cells, cellular debris, and occasional cholesterolin-crystals are found in these cheesy masses. In older, darker-hued masses an offensive odor is given off, and numerous micrococci and bacteria are found. In adults, calcareous infiltration of the cheesy little masses may be met with. *Parenchymatous tonsillitis* is shown by a greater enlargement of the tonsil, due to a marked infiltration of all the tissues. Suppuration in the tonsil is frequent, the follicles usually bursting and uniting in abscess-formation. Pus may burrow into the cellular tissue surrounding the tonsil, and find its way even down to the clavicle. The *herpetic or ulcero-membranous form* of tonsillitis described by Rilliet and Barthez, DaCosta, and others, in which an eruption of herpetic vesicles on the tonsils is followed by their rupture and the formation of a lightly adherent, membranous covering, is rarely met with. It is said to complicate *superficial* or *catarrhal tonsillitis* (Allechin). In *necrotic tonsillitis* (Strümpell) a grayish-white adherent necrotic membrane is observed, that is limited by the inflamed membrane surrounding the mucosa covering the tonsils, which are moderately swollen. After the removal of the slough a dirty and often deep ulcer remains.

**Etiology.**—Predisposing causes are age, sex, temperament, and atmospheric conditions. The disease is more common in youth and in early adult life than during infancy, although enlarged tonsils are frequently met with at this period of life also. Boys and young men appear to be attacked more often than the opposite sex. An individual susceptibility is most distinct in lymphatic and strumous constitutions;



this may be simply hereditary, or in certain cases it is aggravated by or tends to recur especially in the rheumatic diathesis. It is certain also that one attack of acute tonsillitis predisposes to subsequent ones, particularly when the first attack has left some enlargement or hypertrophy of the tonsils. Sudden, rapid, and extreme climatic changes, and the special local and atmospheric conditions sometimes seen in connection with outbreaks of scarlet fever, measles, and diphtheria, have an undoubted influence predisposing to epidemic tonsillitis.

The *exciting causes* of acute tonsillitis are most commonly the following: (a) exposure to cold and dampness, or talking in a cold, moist atmosphere; (b) exposure and talking in an overheated atmosphere vitiated with smoke or other poisonous and irritating vapors or gases; (c) bad drainage, sewer-gases; (d) specific infectious fevers, as scarlatina, measles, and erysipelas; (e) irritation from hard and sharp foreign bodies or chemical irritants; (f) the presence of microbes.

**Clinical Symptoms.**—Three principal varieties of acute tonsillitis occur clinically, the symptoms of which will be described under their respective headings:

(a) **Acute Catarrhal or Superficial Tonsillitis.**—This form is often associated with acute pharyngitis. The earliest *local symptoms* are pain and difficulty in swallowing, the former often becoming quite acute and radiating to the ear and lymphatics at the angle of the jaw, where tenderness on pressure may also be elicited. In speaking a nasal twang is often noticed. During the laborious act of swallowing the sensation of a lump in the throat, especially when the mouth is dry, is commonly complained of. Simple stomatitis, with its discomforts, may be associated, and rarely there is a slight cough with the painful expectoration of a sticky mucus which accumulates in the throat (Browne). There may be salivation, and usually there is a bad taste in the mouth, with fetor of the breath. *Inspection* shows the tonsil to be red and swollen. Though dry and glazed at first, the surfaces soon become covered with a thin exudate of muco-pus, which is easily detached by brushing, gargling, or “hawking” the throat. There is usually some accompanying redness, and also a *tumefaction* of the uvula and faucial pillars. The *constitutional symptoms* of simple erythematous tonsillitis at the outset are mildly febrile. The attacks usually come on rapidly, and last but a few days, subsidence taking place rapidly also. *Otitis media* may follow the extension of the tonsillar inflammation, and *acute pharyngitis* is a more common complication; endocarditis and pericarditis rarely also occur.

(b) **Acute Lacunar or Follicular Tonsillitis.**—In this form, which is quite common in children, not only the mucous membrane lining the crypts is inflamed, but that covering the surface of the tonsils also, giving rise to more or less associated *catarrhal tonsillitis*. The *local subjective symptoms* in this disease are pain, tenderness, and difficult deglutition, the counterparts of those of the preceding form. The tonsils are seen to be covered with small, slightly prominent, whitish-yellow spots or patches of a characteristic creamy exudate corresponding to the position of the crypts and numbering from two to eight or ten or more. These little masses or plugs may be pressed out of the follicles with a spatula. A predominance of pus-cocci and cells may rarely forerun the further formation of little follicular abscesses, and even of slight erosions

and ulceration of the mouths of the lacunæ. Unlike simple catarrhal tonsillitis—at least in so far as simultaneous involvement is concerned (Cohen)—both tonsils are usually affected in this trouble, though one to a greater degree than the other. The whole tonsil is considerably swollen, and in severe cases the cervical lymph-glands also. The *constitutional symptoms* of follicular tonsillitis may be quite severe. The disease may be ushered in with a pronounced chill, headache, aching of the back and limbs, marked anorexia, a heavy deposit of urates, and insomnia, along with a rapid rise in the temperature to 103° or 104° F. (39.4°–40° C.)—in children as high as 105° F. (40.5° C.). The general depression may be so great as to simulate adynamia. Though sudden in its onset and rapid and often intensely acute in its progress, the disease seldom lasts more than five or eight days. Follicular abscesses, febrile albuminuria (Hais-Brown), may appear and complicate the case, while chronic swelling of the tonsils, desiccation, and bacterial degeneration of the lacunar masses may be sequelæ. The latter give rise to an offensive odor when crushed, and often cause hypochondriac patients needless anxiety by being mistaken for tuberculous deposits. The retained follicular exudates may undergo calcification, and may be expectorated along with concretions or chalk-plugs.

(c) **Acute Parenchymatous Tonsillitis** (*Tonsillar Abscess* or *Quinsy*).—In this form of tonsillitis, which occurs most often during adolescence and early adult life, the symptoms reach the most pronounced and severe types. The stroma is inflamed and the tendency is toward suppuration.

*Local Symptoms.*—Complaint is first made of dryness of the throat, with painful and difficult deglutition. The pain is a prominent subjective sign, and may be referred to one or both ears according as one or both tonsils are inflamed. The secretion of a viscid mucus soon takes place, and as the tonsillar swelling increases, the husky voice of sore-throat and difficult articulation supervene; in cases of aggravated swelling dyspnea may often appear later. On examining the tonsils they are found to be greatly enlarged, deeply reddened, and edematous. The surrounding soft parts, the faucial arches, pillars, and the uvula, also manifest a deep congestion. The swollen tonsils may cause a bulging forward of the anterior pillars of the fauces, and push the often elongated and edematous (jelly-like) uvula to one side; or if both tonsils are affected, they may grasp or push it forward. In severe cases the tonsils may meet in the median line. They are firm to the touch. Patches showing follicular tonsillitis are not infrequently seen associated with the trouble. The submaxillary glands may be engorged, and opening the mouth is often performed with difficulty; it is usually only partial, on account of the fixation of the jaw.

In a few days, perhaps, softening and fluctuation may be detected in the tonsils, and spontaneous rupture and discharge of the pus may occur, with almost instant relief to the patient. Suppuration and tonsillar abscess are not always the termination, however, of parenchymatous inflammation, resolution sometimes taking place in the milder cases. The abscess may open in one or more places, and rupture during sleep may rarely cause suffocation by the entrance of pus into the larynx. The tonsil may regain its original size in a few days after the discharge of pus, and all the symptoms subside. The *constitutional phenomena*

of parenchymatous tonsillitis are usually severe from the start, even in children, and more so than in the follicular form (Mackenzie). The temperature rises to 104° or 105° F. (40° or 40.5° C.), and the pulse-beats may reach 130 per minute. The usual symptoms accompanying inflammatory fever are marked. There may be delirium, and the symptoms generally increase until the abscess bursts or is opened, when the constitutional as well as the local disturbance rapidly abates.

*Course, Duration, and Terminations.*—Though often severely acute in its course, quinsy seldom goes on to rupture in children, usually ending in resolution in from three to five days. If both tonsils are inflamed, only one suppurates as a rule, or but one at a time. The duration of an attack ending in tonsillar abscess is about eight or ten days in adults.

*Complications and Sequelæ.*—The tonsillar suppuration may invade the cellular tissue between the tonsil and the pterygoid muscles; a peritonsillar abscess may then result that may open even above the clavicle. Deep involvement of the tonsil may also cause ulceration into the internal carotid or internal maxillary arteries, and fatal hemorrhage occur, though these accidents are, fortunately, rare. Edema of the larynx is also an infrequent complication. French writers, as Guble, Germain Sée, and others, have reported cases of paralysis of the soft palate and pharynx following inflammatory throat-diseases. On subsidence of the tonsillar inflammation the trouble becomes evident in the difficult swallowing and partial regurgitation of liquids and solids into the nasal passages, and in the nasal intonation of the voice. A frequent sequel, especially in those predisposed by heredity, is chronic enlargement of the tonsils.

(d) **Necrotic Tonsillitis.**—This affection is considered by Strümpell to be in some instances entirely distinct from diphtheria in its etiology, although he admits that quite frequently it is simply a mild form of the latter disease, and that often it is impossible to distinguish between the local appearances of the two conditions: these have been referred to under the heading of Morbid Anatomy. The constitutional disturbances are severe, especially in children, though they seldom last longer than a week, and are followed by a rapid convalescence. The cervical glands are not swollen to the same extent as in diphtheria. The occurrence later of palatal and pharyngeal paralysis in a supposed case of necrotic tonsillitis would point to its true diphtheritic origin.

**Diagnosis.**—The appearance of the several forms of acute tonsillitis, associated with the clinical history of each case, should enable a ready diagnosis to be made in the majority of cases. A difficulty may, however, arise in discriminating follicular tonsillitis from diphtheria, especially when the pseudo-membranous exudate of the latter is limited to the tonsils. The appended table gives the important points of differentiation between these diseases:

#### FOLLICULAR TONSILLITIS.

A soft, pultaceous, yellowish-white deposit occurs in spots or patches situated over the mouth of the follicles, with areas of redness intervening.  
The exudate is easily removed, leaving a smooth surface.

#### DIPHTHERIA.

A tough, ashy-gray, continuous, and uniform pseudo-membranous deposit covers the tonsils.  
Very adherent, and can be torn off in strips only, leaving a bleeding erosion.



## FOLLICULAR TONSILLITIS.

The deposit is limited to the tonsils (important).

If the creamy deposits unite to form a continuous layer, removal is either not followed by re-formation, or very late.

May have high temperature, but lasting only a day or two. Albuminuria extremely rare, if present at all.

Cervical lymphatic glands seldom or slightly swollen.

Complications rare and mild.

Cellular detritus, bacteria, etc. in deposit.

## DIPHTHERIA.

The pillars of the fauces and uvula are involved as well.

Removal of the membrane is followed by re-formation within twelve to twenty-four hours.

Persistent elevation of the temperature; more or less albuminuria is common.

Usually markedly swollen glands.

Complications frequent and grave.

Fibrinous exudate, etc. containing the Klebs-Löffler bacillus.

Cases seen early, with severe constitutional symptoms and red and *swollen tonsils* having no deposit, may give rise to the question whether *simple angina* or *scarlet fever* is to follow. In such cases the latter disease may be excluded by a negative history of exposure to contagion, by the absence of a very high pulse-rate, and by the non-appearance of the scarlatinal eruption. Necrotic tonsillitis may be discriminated from the lacunar variety in the same manner as diphtheria—*i. e.* by its local manifestations, a full description of which has already been given under the heading of Morbid Anatomy.

The **prognosis** is good as regards life, and favorable as regards complete recovery. The occurrence of either fatal hemorrhage or asphyxia in *quinsy* is extremely rare. In debilitated and strumous individuals relapses are prone to occur, and successive acute attacks of tonsillitis tend to cause permanent hypertrophy of the tonsils. In cases of *necrotic tonsillitis*, especially during the earlier periods, the prognosis should always be guarded.

**Treatment.**—Particularly in the lacunar and necrotic forms of tonsillitis the patient should be kept apart from others as much as possible, since both types appear to be contagious to a certain degree; or, if other persons in the house are subject to a common source of infection—not human—their frequent nearness to a given case only serves to augment their own liability to similar attacks. Individual susceptibility to frequent attacks of sore throat may be lessened by systematic cold bathing of the neck. Constitutional and local rest is a first and constant requisite. Efforts at swallowing and talking should be reduced to a minimum, and in marked cases of follicular or suppurative tonsillitis rest in bed is often sought without direction. Bland nourishing liquids, as milk, broths, and the like, should constitute the only nutriment during the stadium of the tonsillar inflammation. Early in the case a free evacuation of the bowels should be obtained, and small doses of calomel (gr.  $\frac{1}{8}$ — $\frac{1}{6}$ —0.008.—0.010, repeated hourly until about gr. 1—0.0648—has been taken), followed by a Seidlitz powder or Rochelle salts in hot water, will be effective in most cases. In severe cases of quinsy relief from the pain is urgently called for, and either a Dover's powder or a hypodermic injection of morphin (gr.  $\frac{1}{6}$ — $\frac{1}{4}$ —0.010—0.016) and atropin (gr.  $\frac{1}{100}$ —0.0006) will probably suffice for their relief. A high temperature must be combated by small doses of aconite, frequently repeated: this drug has been much used in the follicular ton-

sillitis of children. Quinin, in solution with dilute sulphuric acid, is also often given.

The administration of sodium salicylate or benzoate, of salol, or of the ammoniated tincture of guaiac in 1-dram (4.0) doses (Sajous), seems to lessen the duration and severity of tonsillitis, and even to cure some cases of the lacunar form within forty-eight hours and without local applications. The tincture of the chlorid of iron in glycerin (4 or 5 drops to the dram—4.0—given every two hours) is regarded by Bosworth as almost specific at the commencement of an attack of acute follicular tonsillitis. During convalescence semi-liquid and soft, light foods may be allowed gradually; and bitter tonics and iron are to be administered if there are depression and anemia. The following is a favorite prescription:

R <sub>y</sub> . Strychninæ sulph.,	gr. ss (0.032);
Syr. acaciæ,	℥ss (16.0);
Liq. ferri et ammon. acetat.,	q. s. ad ℥iij (96.0).—M.

Sig. 3j (4.0) *t. i. d.*, in water, after meals.

If the case is seen quite early during congestion, the use of cold is of great value in giving local relief and in shortening the attack. Ice may be sucked, and flannel dipped in ice-water and wrung out may be applied around the neck, or an ice-bag used. Lozenges of guaiac (gr. ij—0.129) or the ammoniated tincture in 1-dram (4.0) doses in milk, and used as a gargle, are indicated early, and, according to Sajous, seldom fail to control or arrest the inflammation. Equal parts of the tincture of the chlorid of iron, glycerin, and water, applied gently with a camel's-hair brush, have long been used locally on the surfaces of the tonsils, and with marked benefit. Alkaline and mild antiseptic solutions, used as gargles or sprays (preferably the latter), are generally useful. Thus, Dobell's solution, or Seiler's tablets dissolved in water, or borax and thymol, or carbolic acid, or potassium permanganate in weak solution, may be serviceable. Mild counter-irritation at the angle of the jaw by means of iodine or slightly irritating embrocations is helpful.

Early scarification of the tonsils as a depletory measure, and painting with cocain (10 per cent.), I have found useful to bring about resolution.

Astringent sprays containing alum or silver nitrate are often efficacious after a day or two. When the case is first seen and fully developed, the atomization of a warm solution of cocain (4 to 8 per cent.) or lime-water, with the external application of heat by means of poultices, is indicated. Should gargling be possible, nothing is better than hot water or milk. If, in parenchymatous tonsillitis, fluctuation be detected or suppuration be even suspected of commencing, the prompt use of the bistoury (the blade being guarded by wrapping with cotton or adhesive plaster), with the production of free bleeding or the discharge of pus, will give great satisfaction and relief. The patient's head, especially if it be a child, should be tilted forward during the operation, so as to allow most of the blood and pus to pass into the mouth. When incision of the tonsil fails to bring pus, it has been advised to puncture through the anterior pillar, where pus may be formed in the cellular tissue in front of or behind the tonsil.

When the tonsillar enlargement threatens life through suffocation,

excision of the tonsils, laryngotomy, tracheotomy, or intubation may have to be performed.

## CHRONIC TONSILLITIS.

(*Hypertrophied Tonsils; Adenoid Vegetations.*)

**Definition.**—Enlargement of the tonsils (faucial and pharyngeal), due to chronic inflammation or hypertrophy, and usually associated with or causing a perverted local and systemic condition.

**Pathology.**—The faucial tonsils show a true chronic hypertrophy of the lymphoid and fibrous elements. According to the hyperplasia of the latter the organs will be smaller and more indurated. They may be rough on the surface from “distended lacunæ or ruptured follicles” (Berkley Robinson), the latter being in a state of chronic inflammatory thickening, and showing caseous degeneration of their contents. The growths in the vault of the pharynx are adenomatous papillomata; they are either sessile or pedunculated, and are fleshy in appearance and consistence and very vascular. They range in size from a grain of wheat to an almond-kernel (Allen), and project from the pharyngeal vault, lying in the depression posterior to and on a line with the fossa of the Eustachian tube (Rosenmüller’s fossa). “Hypertrophy of the pharyngeal adenoid tissue may also be present without great enlargement of the tonsils proper” (Osler). A congestive type of nasal catarrh in adults often accompanies, or is the result of, neglected adenoid growths and hypertrophied tonsils that date from childhood. Chronic pharyngitis is also not infrequently associated.

**Etiology.**—The *predisposing causes* of chronic hypertrophy of the tonsils are—(a) heredity, especially in the scrofulous and syphilitic diatheses; (b) age, most frequently between five and fifteen years; (c) sex, boys appear to be affected more frequently; (d) hygienic surroundings.

The *exciting causes* are usually previous attacks of acute tonsillitis, either simple or that which is symptomatic of diphtheria or scarlatina. According to Harrison Allen, adenoid growths from the normal lymphoid tissue of the vault of the pharynx (pharyngeal tonsils) may be congenital, and are “in some way associated with the canal which is found in early fetal life penetrating the brain-case and uniting the anterior part of the pituitary body to the lining membrane of the pharynx.”

**Symptoms.**—*Local.*—With slight or even moderate tonsillar enlargement there may be few or no symptoms attributable to it. There may be simply an increased secretion of mucus, and a susceptibility to fresh anginal attacks or to severe tonsillar manifestations in diphtheritic or scarlatinal attacks.

The first symptom to attract the attention is the direct effect of naso-pharyngeal obstruction—*i. e.* oral respiration. This mouth-breathing is visibly labored and abnormally audible, and is especially marked at night, the child’s respiration being noisy, snorting, and irregular. Sleep is disturbed by paroxysms of dyspnea, sometimes due, perhaps, to reflex spasm of the glottis. Nightmare follows as a result of imperfect aëra-



tion of the blood which supplies the brain on account of the obstruction to perfect respiration. The act of swallowing is rendered difficult by the faucial obstruction, and is often painful, owing to the superadded acute tonsillar trouble that is so liable to occur in the hypertrophied glands. Indirect results of chronic tonsillar enlargement are a laryngeal stridor and a croupy cough. Sometimes asthmatic attacks coexist, and seem also to be due to the hypertrophy. An excessive secretion of mucus in the pharynx is a common symptom, and causes hawking in subjects past young childhood. The hearing is often impaired, and tinnitus aurium is complained of, being the result of pressure of the growths against the orifice of the Eustachian tube or of clogging of the tube with mucus, due to the extension of inflammation from the naso-pharynx.

Absolute deafness may result, and the senses of taste and smell are likewise diminished or perverted. The signs of chronic tonsillar enlargement and pharyngeal adenoid growths are interesting and important. Inspection of the fauces will show the tonsils bulging as two lumps covered with thick mucus, or the latter may ooze around the uvula from the pharynx. In mouth-breathers of long standing the superior dental arch is narrowed and the hard palate is highly arched. The breath is fetid, owing to the cheesy, inspissated exudate in the tonsillar crypts. In very old cases a tonsillar calculus may be felt, and is the result of calcification of the secretion.

The facial expression is characteristically stupid and pathetic; the disposition is dull, irritable, and stubborn; the lips are thick, and a vacant stare is in the eyes. Speech is slow, phonation nasal in quality, and articulation of the nasal consonants *n* and *m*, *l* and *o*, is changed or muffled. Stammering is not rarely associated with tonsillar hypertrophy. The anterior nares may be dilated and present a pinched appearance above their openings.

The prolonged interference with normal respiration gives rise to a peculiar chest-conformation, simulating that of rickets (*chicken-breast*). The ribs are prominent anteriorly, and there is a marked forward angle at the manubrio-gladiolar junction, as well as a grooved depression at the ensiform cartilage. Depressions between the widely-separated ribs exist anteriorly also. Posteriorly, and at the base of the chest in particular, the intercostal spaces are practically absent on account of the closeness of the ribs. The upper part of the chest is very narrow and the shoulder-bones quite prominent; the antero-posterior diameters of the thorax are less than normal; the sides are unusually curved.

On percussion the hepatic area of dulness is diminished on the chest-wall, but increased downward and to the left. The first cardiac sound is weak. On inspiration there is a retraction of the intercostal spaces in the lower and lateral thoracic regions. The lymphatic glands of the neck may be moderately swollen.

The *general symptoms* of tonsillar hypertrophy are more marked when the growths exist in the pharyngeal vault alone. Developmental processes in children, such as dentition, and at puberty, particularly when the voice-changes are looked for, are often retarded or perverted. Anemia, headache, especially during study, cardiac palpitation, enuresis, and habit-chorea of the facial muscles, may be associated with general

capriciousness, mental dulness, indisposition to intellectual exertion, drowsiness, and sullen irritability. The term *aproseria* has been given to the loss of power to concentrate the mind for any length of time that is so characteristic of these cases.

**Diagnosis.**—Inspection of the fauces will reveal enlarged tonsils. It should be borne in mind, however, that the act of gagging often causes the tonsils to rotate forward and inward, making them appear larger really than is the case. Adenoid growths of the pharyngeal vault may exist without tonsillar enlargement, and can be detected by posterior rhinoscopy or by the insertion of the finger into the nasopharynx; the vegetations may thus be felt blocking the vault.

**Differential Diagnosis.**—It is important not to attribute the obstructive symptoms to nasal hypertrophies or atresia or to *malignant growths* in the naso-pharyngeal space. The latter are infrequent at the ages at which chronic tonsillar enlargement of the fauces and pharynx is most apt to occur—*i. e.* early in life. Again, palpation of sarcomatous or carcinomatous growths gives marked differences in consistence, and there are usually spontaneous hemorrhages and local pain in attendance upon these neoplasms. “*Thumb-suckers*” differ from mouth-breathers in that in the former the incisors are inclined forward and cause slight protrusion beneath the upper lip; the dental arch is flat. In mouth-breathers, however, the incisors are vertical or nearly so, or incline so as to overlap each other; the dental arch is high and curved (H. Allen). *Retropharyngeal abscess* may be confounded with tonsillar enlargement, especially in children. But in this disease the attacks of dyspnea, the dysphagia, and the local distress are more marked. Again, in the pharyngeal disease the swelling is in the median line, pushing the soft palate forward perhaps, and on palpation it may give a sense of elasticity or fluctuation to the finger. Slight fever may also be present.

**Prognosis.**—Tonsillar hypertrophy is not a severe disease as regards life. There is, however, an increased liability to contract colds, recurrences of follicular tonsillitis, attacks of diphtheria, and severe scarlatinal angina. The prognosis in acute respiratory affections associated with chronic tonsillar enlargement is always more or less grave. Adenoid growths, even when neglected, tend to lessen in size after puberty, with a subsidence of local and reflex symptoms. After removal the growths, as a rule, do not return.

**Treatment.**—The old-fashioned use of astringent applications is probably useless when there is any marked chronic enlargement of the tonsils, and active surgical treatment alone is to be recommended for the condition. The use of absorbents and caustics, either externally or by parenchymatous injection, is, I am inclined to believe, objectionable on account of the necessarily protracted and painful course of treatment.

There are no more satisfactory means of doing radical good in cases of this kind than the galvano-cautery, scarification, and the removal of the tonsils with the tonsillotome, snare, or bistoury. In offensive follicular disease applications of chromic acid may give good results. Adenoid growths may be removed by means of the finger, curet, or forceps.

*Constitutional treatment* is often necessary in improving the nutrition of the patient. Good food, a change of air, systematic bathing, prudent

habits, careful dress, and medicinal tonics and alteratives, as cod-liver oil, iodid of iron, and the hypophosphites, are usually indicated.

## V. DISEASES OF THE PHARYNX.

### PHARYNGITIS.

#### ACUTE PHARYNGITIS.

(*Pharyngitis Acuta Simplex*.)

**Definition.**—An acute catarrhal inflammation of the mucous membrane of the pharynx.

**Pathology.**—The mucous membrane is congested diffusely or in patches, and there may be an inflammatory exudate in, and a consequent swelling of, the submucosa and the contained glandular structures. The surface of the membrane is more or less coated with a viscid muco-pus.

**Etiology.**—Predisposing causes are—age, it being more frequent in adolescence and young adult life; a depraved constitution; digestive disorders; and a rheumatic, gouty, or serofulous diathesis. The usual exciting cause is exposure to cold or sudden changes of temperature and climate and to irritating vapors. An acute naso-pharyngeal catarrh, by bathing the pharyngeal mucosa with its irritating secretions, may set up the trouble. "*Epidemic pharyngitis*" is probably a manifestation of influenza. Acute simple pharyngitis may be a complication of scarlatina, measles, and small-pox (*exanthematous pharyngitis*) and of erysipelas (*erysipelatous pharyngitis*); in the latter disease, moreover, it may become gangrenous or suppurative.

**Symptoms.**—*Locally*, the affection is ushered in with a feeling of dryness and soreness, especially on swallowing. With the production of the muco-purulent secretion a tickling sensation provokes *hawking* or a slight "*throat cough*" and efforts at expuition. The catarrhal process may extend to the larynx and cause some hoarseness, or to the Eustachian tube, causing dulness of hearing. Movements of the neck are *painful* and *stiff*, particularly if there is, as is often the case, slight involvement of the lymph-glands. *Inspection* of the throat shows the pharynx, often the posterior pillars of the fauces and the soft palate, and even the anterior pillars and tonsillar surfaces, to be deeply reddened and tumefied; the coursing veins are enlarged, and particles of a yellowish-white secretion appear here and there. Sometimes the pharyngeal follicles become subject to acute inflammation, and appear as elevated, discrete, shiny spots (*herpetic pharyngitis*—Mackenzie).

At the onset of this affection there may be chilliness, followed by slight fever, headache, an accelerated pulse, a dry skin, and anorexia. The pharyngeal symptoms seldom last more than from three to five days, when resolution takes place, some tenderness of the pharynx, however, remaining for a time.

**Diagnosis.**—On examination of the throat there should neither be any difficulty in diagnosing the affection nor any likelihood of confounding the affection with simple tonsillitis.



The **prognosis** is always favorable. In weakly patients, however, there is a liability to subsequent attacks.

**Treatment.**—In the early stages sucking of small pieces of ice does much to allay the congestion and irritability. A spray of cocain or menthol in alcohol (2 per cent.) may also be used, followed by a 4 per cent. solution of antipyrin; Dobell's solution is always to be recommended for its alkaline, sedative, and antiseptic action. Swabbing the pharynx with a silver-nitrate solution (gr. xl to the ounce—2.59 to 32.0) is, according to Sajous, of great benefit.

When the disease is well established relief is often obtainable by medicated steam-inhalation, as with the compound tincture of benzoin. In rheumatic cases lozenges of guaiac (gr. ij—0.194) are useful. The sipping of hot milk in which sodium bicarbonate has been dissolved is very soothing to the inflamed mucosa.

The general treatment embraces measures directed at the fever and the diathetic condition. A hot foot-bath and a calomel purge, with belladonna, acetanilid, or aconite for the fever and pain, and sodium salicylate (gr. lx-lxxx—4.0–5.1—in the twenty-four hours), may be required. The diet, of course, during the height of the attack, should either be liquid or semi-solid.

Persons susceptible to repeated attacks must exercise caution in regard to exposure to severe cold and weather-changes, irritating vapors, and the like. Daily cold sponge-baths may be used to harden the skin. Tonic, nutrient treatment is also frequently called for.

#### MEMBRANOUS PHARYNGITIS.

(*Pharyngitis Crouposa*.)

**Definition.**—An acute superficial inflammation of the pharyngeal mucosa, characterized by the formation of a whitish false membrane, due usually to the streptococcus.

**Etiology.**—The principal causes of this form of pharyngitis are exposure of persons in debilitated health to cold or an impure or a septic atmosphere, particularly during epidemics of such diseases as scarlatina.

**Symptoms.**—The local and general symptoms are those of ordinary sore throat, though of a more severe type.

**Diagnosis.**—The pseudo-membrane is thin, of a yellowish-white color, and appears in small patches over the pharynx; it is easily detached, and is thus distinguished from diphtheria, with which alone it might be confounded. The presence of small vesicles or ulcers and the absence of grave constitutional disturbances are also features in this affection that serve to differentiate it from diphtheritic pharyngitis.

The **prognosis** is favorable.

**Treatment.**—Local applications of solutions of hydrogen peroxid or potassium permanganate (gr. x to the ounce—0.648 to 32.0) are very satisfactory. For the painful dysphagia the sedative and soothing remedies suggested for simple acute pharyngitis may be used. Internally, sodium benzoate (gr. v-xv—0.324–0.972) in glycerin, elixir of calisaya, and salol have each been recommended. Tonic treatment is nearly always needed.

## CHRONIC PHARYNGITIS.

**Definition.**—A chronic inflammation of the mucous membrane of the pharynx. It may consist of either a hypertrophic or an atrophic involvement of the follicles, or both processes may coexist.

**Varieties.**—(a) Chronic naso-pharyngeal catarrh; (b) chronic hypertrophic pharyngitis or naso-pharyngitis (*pharyngitis sicca*); (c) follicular or granular pharyngitis. The last named is probably the result of, and nearly always is associated with, chronic simple (or hypertrophic) pharyngeal (or naso-pharyngeal) catarrh.

**Pathology.**—The mucous membrane in simple chronic pharyngitis is either reddened, thickened, and viscid (hypertrophic form), or pale, thin, and dry (atrophic form); in both instances dilated and tortuous veins are prominently shown. In the follicular variety the pharyngeal mucous glands are swollen into little red, glistening nodules studding the congested membrane. The enlarged follicles are due to a hyperplasia of lymphoid cells and an accumulation of retained dried-up secretions.

**Etiology.**—A protracted impairment of the general health, especially in those who over-exert mentally and are of sedentary habits, is a common predisposing cause of chronic pharyngitis. Repeated acute attacks may precede the affection, or it may develop subacutely and almost imperceptibly. It is most common in adolescent and middle life.

The exciting causes are frequent and prolonged over-use and strain of the voice in clergymen, singers, teachers, army-officers, and street-venders; irritation from tobacco-smoke, chemical vapors, and continued exposure to cold air; and perhaps the persistent swallowing of very hot or cold foods, stimulants, or spicy articles.

**Symptoms.**—In all varieties of chronic pharyngitis the *local discomfort* is often very slight, and more annoying than painful, except when an exacerbation takes place. It is a particularly uncomfortable condition in those whose occupation requires more or less constant use of the voice. There is a sensation of *dryness* and *tickling* or *burning* in the throat and the desire to clear the throat of sticky mucus by *hawking* or a *short cough*. These symptoms are usually worse on rising in the morning, especially if some unfavorable influence has been exerted during the night previous, the throat being dry and a viscid secretion having collected. Swallowing is seldom interfered with.

If the larynx is somewhat affected by extension of the pharyngeal inflammation, *hoarseness* and a *dry, hacking cough* are produced. After using the voice there is a sense of fatigue, with huskiness and often some irritability.

The *local appearances* of chronic pharyngitis vary according to the form of the affection present in the case. In chronic catarrh of the pharynx a considerable collection of mucus is seen adhering to the mucosa and extending downward from the posterior nares. The senses of hearing and taste may be impaired. The uvula is frequently elongated, and its tip may rest on the base of the tongue. A nasal intonation of the voice is sometimes provoked. The posterior nares as seen by the rhinal mirror are often stopped up by foul secretions or by hypertrophy of the nasal mucous membrane. Headache and attacks of vertigo may occur.

*Chronic hypertrophic pharyngitis* and follicular pharyngitis ("clergyman's sore throat") are commonly associated. The thickened, reddened, pimply, vein-coursed appearance of the mucosa is characteristic. The follicles may be seen sometimes as polypoid elevations, and the pharyngeal tonsil may be found by the finger to be enlarged (Kölliker).

In the dry, *atrophic pharyngitis* that occurs more often in later life, and as a sequel of the simple chronic or follicular variety, a pale, smooth, relaxed, lustrous, and often quite painful membrane is observed.

The *general symptoms* are usually those of a weak, debilitated, nervous constitution, though in mild cases the general health may be unimpaired. In atrophic pharyngitis considerable cachexia may be present.

**Diagnosis.**—Care should be exercised in discriminating the variety of chronic pharyngitis present in any given case, so that the treatment may be planned accordingly. Careful and repeated inspection of the throat must render the diagnosis easy unless ulceration has taken place: in such cases a tuberculous or syphilitic sore throat must be eliminated by the superficial character of the ulcers, by their ready response to proper treatment, by the history of the case as to specificity, and by the absence of marked pain or constitutional or pulmonary symptoms pointing to tuberculosis.

**Prognosis.**—This should be guarded as to cure, on account of the stubborn resistance to treatment and the difficulty in removing unfavorable influences. Acute exacerbations are liable to recur unless rigid prudence and caution are practised at all times in avoiding the cause of the trouble.

**Treatment.**—The local use of astringent and alkaline antiseptic sprays or of the nasal douche is usually recommended, but has only a palliative effect. Silver-nitrate cauterization may be tried. The only effectual means, however, of curing the follicular or hypertrophic variety is that used by most throat-specialists—namely, the wire galvano- or actual cautery. Applications of silver nitrate (gr. x to the ounce—0.648 to 32.0) and the internal use of the oleoresin of cubebs have been recommended for the atrophic pharyngitis. Insufflation of powdered tannin or alum is also of service.

*Systemic disturbances* need attention according as they present themselves. Mineral baths are sometimes of great benefit, and tonics are usually indicated. It is of prime importance that all irritating causal factors be removed or avoided before any favorable results can be hoped for from local applications. Tobacco-smokers and toppers must deny themselves their habitual luxuries. Krause and Heryng recommend with favor curetting and the application of lactic acid to superficial tuberculous ulcers.

## ACUTE INFECTIOUS PHLEGMON OF THE THROAT.

**Definition.**—An inflammation of the pharyngeal mucosa that passes rapidly into a suppurative process.

Its **etiology** is not definitely known. I have met with no cases except in my hospital wards, though they doubtless occur in general medical practice. The clinical features have been described by Senator.

The **symptoms** are *sudden in their onset* and quite intense. They



are severe soreness of the throat, dysphagia, and hoarseness, as a rule; in advanced cases there has been difficult respiration. *Inspection* shows the pharynx to be deeply injected and the seat of marked inflammatory edema, the neck appearing greatly swollen as well. The general disturbance is correspondingly severe.

The **treatment** is wholly symptomatic.

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### RETROPHARYNGEAL ABSCESS.

**Definition and Pathology.**—A suppurative inflammation of the connective tissue lying anterior to the cervical spinal column.

The disease is rare, though it is relatively most common before two years of age. It is usually a primary affection, occurring without assignable cause, but a certain proportion of instances are doubtless caused by caries of the cervical vertebræ. It may rarely be secondary to any of the specific fevers.

The **symptoms** are *pain* in swallowing, *impeded respiration*, soon becoming stertorous in character, the dyspnea meanwhile constantly increasing. There may be *cough*, and the voice may present abnormal characteristics. The signs of *stenosis* finally declare themselves with considerable violence, and an examination of the pharynx usually serves to make the diagnosis positive; the projecting tumor is visible, and the palpating finger readily detects fluctuation. In infants, however, this procedure may be attended with great difficulty.

The **course** of the disease may be acute, lasting one or two weeks; more frequently, however, it is subacute (rarely chronic), as, for example, when it is due to caries of the vertebræ.

The **prognosis** is favorable in all cases that are early and properly diagnosticated. If unrecognized until the later stages have been run, suffocation may ensue, or in the event of spontaneous rupture pus may pour into the larynx and cause death by asphyxia.

**Treatment.**—As soon as fluctuation is detected the abscess should be freely opened, and preferably, as a rule, through the mouth by means of a guarded bistoury. The throat, after the abscess is thoroughly evacuated, should be washed out with some mild antiseptic solution (salicylic acid 2 per cent. or boracic acid 2 per cent.). When pointing occurs at the side of the neck, as sometimes happens, the incision should be made through the skin in that locality. Constitutional indications are to be fulfilled in accordance with general principles, and the strength of the patient is to be maintained by a highly nutritious dietary.

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## VI. DISEASES OF THE ESOPHAGUS.

### ESOPHAGITIS.

#### ACUTE ESOPHAGITIS.

**Definition.**—An acute inflammation affecting either the mucous membrane or submucous tissues of the esophagus, or both.

**Pathology.**—The ordinary morbid changes of an acute esophagitis

are those of a simple catarrhal inflammation of the mucosa. It is rather characteristic of the condition that there is no increased secretion, a sponginess and rapid desquamation of the epithelium taking place instead, and causing a granular appearance of the membrane. Occasionally the mucous glands are swollen, and may break down, with the formation of small follicular ulcers. Catarrhal erosions may also be seen here and there. A croupous or diphtheritic exudate is seldom found in the lower portion of the esophagus, and small-pox pustules are rarely, if ever, seen. A diffuse or circumscribed purulent inflammation of the submucosa may dissect up the mucous membrane so as to considerably diminish the esophageal caliber; pus is usually discharged into the tube. In severe cases of poisoning (*corrosive esophagitis*) sloughing may extend into the muscular layer, and may produce a foul, dark, hemorrhagic mass. A fibrinous cast of the gullet has been vomited up by an hysterical woman (Birch-Hirschfeld).

**Etiology.**—The causes of acute esophagitis, other than traumatic, are rare. Under the latter are included the *mechanical, thermal, and chemical* irritants, such as the presence of foreign bodies and the swallowing of hot liquids, corrosive poisons, “concentrated lye,” mineral acids, and arsenic. The condition may also be the result of the following: (*a*) an extension of catarrhal inflammation of the pharynx; (*b*) specific infectious fevers, as typhoid, typhus, and pneumonitis; (*c*) diphtheria (*pseudo-membranous esophagitis*) by the extension of pharyngeal diphtheria; (*d*) small-pox, giving rise to a pustular inflammation of the gullet; (*e*) local disease, as carcinoma of the esophagus, glandular or vertebral abscess, or laryngeal perichondritis (Strümpell).

**Symptoms.**—*Pain* during deglutition may be referred to the region of the esophagus, and a steady, dull pain may exist beneath the sternum. *Dysphagia* and *regurgitation of food* may be caused by spasm in severe cases. Mucus, blood, and pus may be discharged later. The absence or mildness of pain is not a true indication of the gravity and extent of esophageal inflammation.

**Sequelæ.**—Simple catarrhal or follicular ulcers may appear, and the necrotic form of the disease may be followed by suppurating ulcers, which, if healing takes place, may cause cicatricial stenosis.

**Diagnosis.**—This may be based upon the localization of pain, especially during deglutition; upon the pain occasioned by the passage of the esophageal sound; and upon the mucus, blood, or pus adherent to its bulb on withdrawal, provided carcinoma at the cardiac orifice of the stomach can be excluded. The expulsion of a pseudo-membrane (diphtheritic) from the gullet should be differentiated from esophagomycosis (thrush), especially in children. The diagnosis of the particular form of esophagitis will depend upon the facts elicited relating to the etiology.

The **prognosis** is good in mild cases, and should be guarded in those associated with grave disease. Death may occur in either the purulent or necrotic form.

**Treatment.**—This is entirely symptomatic, and in severe cases is of little value. A soft, bland diet, preferably of milk, may be borne in ordinary instances; if not, rectal alimentation should be resorted to. For the mild cases swallowing of bits of ice, and later of warm demul-

cent drinks, should be recommended. In cases of marked pain and esophageal spasm relief may be afforded by a hypodermic injection of morphin and atropin.

#### CHRONIC ESOPHAGITIS.

Chronic catarrh of the gullet may result from continued irritation by the causes of the acute form, and also from passive congestion due to hepatic cirrhosis, chronic cardiac or renal disease. The last-named conditions may also cause varicose esophageal veins, and fatal hemorrhage may result therefrom. The increased mucous secretion may cause eructations and nausea.

*Postmortem* evidence of esophagitis, either acute or chronic, is found with extreme rarity.

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#### ULCER OF THE ESOPHAGUS.

THIS is a consequence of a simple or follicular catarrh of the gullet or of gangrene. "Catarrhal erosions" and follicular ulcers may occur in numbers, and necrotic ulcers may occur in bedridden persons opposite the cricoid cartilage. The extensive purulent ulceration following the separation of necrotic sloughs may heal and cause contraction and marked stenosis of the tube. Ulcers simulating those occurring in the stomach (*ulceres ex digestionē*) may sometimes be found at the lower end of the esophagus. *Postmortem* digestion, however, must not be mistaken for peptic ulceration. There may be pain or localized points of tenderness on the passage of the esophageal bougie, with some pus and blood on the bulb after its withdrawal. Rest from swallowing should be secured as far as possible. The sipping of hot milk may be soothing, and the slow swallowing of mild boric-acid and sodium-bicarbonate solutions, or of glycozone, may be tried with benefit.

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#### CARCINOMA OF THE ESOPHAGUS.

THIS is the most frequent affection of the tube, and, as it is the commonest cause of stenosis, it is important from a diagnostic standpoint.

**Pathology.**—Carcinoma of the esophagus is primary and of an epitheliomatous nature, the mucous membrane here being composed of pavement-cells. The new growth affects the mucosa first, and then, increasing in size and causing ulceration, it may involve the entire circumference of the tube like a ring-like mass. This may either be hard, dense, and fibrous, or soft and jelly-like. The esophageal lumen is markedly diminished as a rule, although disintegrating ulceration or "flat" carcinoma may encroach upon the caliber but very little. There may be a diffuse dilatation of the esophagus above the growth, as well as an hypertrophy of the circular muscular fibers. The cancerous tumor is found most commonly in the middle and lower thirds of the esophagus.



**Etiology.**—The *predisposing causes* of esophageal carcinoma are age and sex, males past forty years of age being the usual subjects of this neoplasm. The *exciting causes* are of uncertain origin. It has been alleged that various forms of protracted irritation of the mucous membrane may cause the development of carcinoma; and especially has this point been maintained in connection with the frequent occurrence of carcinoma of the gullet in toppers. It is also believed by some that as gastric carcinoma may develop from the scars of old ulcers, a like condition in the esophagus may act as a nucleus for a carcinomatous growth.

**Symptoms.**—*Dysphagia* is the earliest symptom of esophageal carcinoma with beginning stenosis of the tube. This gradually and steadily increases, so that liquids alone can be swallowed, and later regurgitation even of liquid foods takes place. There may be considerable *pain*. I recently saw an instance with Dr. W. Frank Haehnlen in which enormous quantities of mucus were regurgitated and in which the symptoms of bronchiectasis developed near the close.

The *ejecta* may contain cancerous fragments, blood, and mucus. The dysphagic symptoms may subside spontaneously, owing to the disintegration and ulceration of the growth, or the dysphagia may be so slight as to be masked by the prominent symptoms of hepatic or pulmonary carcinoma and gangrene secondary to a very flat esophageal carcinoma. Or, without secondary manifestations of such a growth, the esophageal symptoms may rarely be latent. The cervical glands may be enlarged.

The most important *general symptom* of esophageal carcinoma, as of this malignant growth elsewhere, is the progressive emaciation, which increases with the stenosis and obstruction to the entrance of nourishment into the stomach. Though seemingly anemic, the patient's blood may contain an excessive number of corpuscles in a given bulk. This is due to inspissation from failure to absorb water and food into the body.

**Course, Duration, and Termination.**—The disease is chronic, becoming progressively worse, and is often beset with grave complications (*vide infra*). It seldom lasts longer than one and a half years, and the duration of medullary carcinoma of the gullet is usually much shorter. A fatal ending is inevitable, by inanition and exhaustion, or as the result of metastasis and secondary complications.

**Complications.**—These follow extension of the cancerous growth to neighboring parts. Thus, involvement of the larynx, trachea, and bronchi has been noted. The cancerous ulcer may also perforate the pleura, the pericardium, or the aorta or its branches, and cause fatal hemorrhage. The vertebrae have been eroded, and compression of the cord, with resulting paraplegia, may take place.

Paralysis of the vocal cords may be the effect of pressure by the growth upon the recurrent laryngeal nerve: most frequently pulmonary gangrene is due to perforation of the lung or to the inspiration of cancerous and decomposing particles that have been regurgitated.

**Diagnosis.**—As the dysphagia is a symptom of paramount importance in the diagnosis of esophageal carcinoma, all other causes of the symptoms must be excluded. Thus, enlarged tonsils, pharyngeal tumors, pressure from without by cervical intrathoracic tumors, as aneurysm, or by displacement of the sternal end of the clavicle, and the presence of foreign bodies or cicatricial strictures of the gullet,—all figure in the production of difficult deglutition. The history of the case,

the age of the patient, the progressive emaciation (cancerous cachexia), and the obstinately increasing dysphagia will enable us to exclude the other affections named. In using the esophageal bougie for diagnostic purposes great care should be exercised, as an aneurysm may thus be ruptured or a deeply ulcerated carcinoma perforated. The withdrawal of cancerous tissue upon the bulb will decide the case. The esophagoscope may be useful in certain cases, but requires great care and special skill.

The **prognosis** is hopeless, and the supervention of grave complications or pulmonary gangrene renders the chances of an early demise very probable.

**Treatment.**—This is essentially symptomatic and sustentative. If feeding by the mouth is difficult on account of the extreme stenosis, although permitting the passage of an esophageal tube, the latter may be used for the passage of liquid nourishment. Rectal feeding may later become imperative. The mechanical treatment of the cancerous stricture by the passage of the graduated esophageal bougie is seldom of any avail, although temporary improvement may perhaps be obtained. Soft, disintegrating, and ulcerating carcinoma should thus be treated, though with the absence of any force whatsoever, lest perforation take place. The performance of esophagostomy may prolong life in some cases.

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## RUPTURE OF THE ESOPHAGUS.

THE first recorded case of this rare condition occurred under the observation of Boerhaave in 1724 in the person of the Baron Wassemar.

**Pathology.**—Softening, together with a great friability, of the esophageal walls may be found, this probably being the effect produced by the solvent action of the gastric juice upon the mucous membrane at a time when the local circulation is disturbed and the vitality of the tissues thus lessened.

The *postmortem* evidence of this accident consists of a longitudinal (as a rule) tear about 5 cm. (2 inches) in length, and situated in the lower half of the esophagus. Food and air may be found to have escaped into the left pleural cavity, and unless death occur at an early date signs of secondary purulent inflammation will probably be noticed. *Postmortem* digestion of the esophagus is more common (Osler). The perforation is often quite large, and is located in the posterior wall of the tube.

**Etiology.**—Softening of the walls of the gullet (*esophago-malacia*) is suggested by Zenker as a condition that always precedes spontaneous rupture, so called. The exciting cause is believed to be violent and persistent vomiting after a particularly heavy meal or during acute alcoholism.

**Symptoms.**—These come on *suddenly* or soon after a full meal, and commence with *nausea* and very *severe vomiting*, accompanied by *great pain* and rapid and extreme *collapse* of the whole body, due to the shock. A cutaneous emphysema of the neck and chest is manifested soon after the rupture.

The **diagnosis**, if made at all, must rest upon the clinical history. Death usually takes place in a few hours, or days at the most, and the **prognosis** is necessarily hopeless.

The **treatment** is equally so in the present status of surgery. Pain, if excruciating, should be dulled by the hypodermic administration of morphin.

## NEUROSES OF THE ESOPHAGUS.

### MUSCULAR SPASM.

(*Esophagismus*.)

**Definition.**—A spasmodic contraction of the muscular layer of the esophagus.

**Etiology.**—It is almost always a secondary affection, met with not infrequently in hysteria, hydrophobia, and rarely in chorea and epilepsy. I have seen one instance of the idiopathic form of the disease in a female possessing a highly neurotic constitution. In this case the esophageal bougie could be passed only with a great deal of difficulty during the spasm: when this relaxed, the bougie glided into the stomach without meeting with any noticeable resistance. It has usually been observed in aged males, and especially in those suffering from hypochondriasis. It may be due to reflex causes, originating, for example, in the uterus; thus, in some cases, it occurs only during the pregnant state.

**Symptoms.**—The chief subjective characteristic is *dysphagia*. Although liquids can be swallowed, solids, as a rule, cannot. Post-sternal *pain* is sometimes noticed, and choking signs are quite common. In the hysteric variety emotional disturbances are found among the prodromata, and most probably bear a causal relation.

**Diagnosis.**—The etiologic factors must be carefully weighed in connection with the symptoms and the valuable testimony gained by the use of the sound. The bougie on reaching the constriction is rather tightly gripped, though gentle pressure soon causes it to relax. After the subjective symptoms and spasm are over the sound passes without the slightest difficulty, providing a point of the greatest diagnostic import. The elderly hypochondriac is, as before stated, liable to develop a similar condition, which must not be confounded with true cancerous stricture.

The **prognosis** is good.

The **treatment** is directed to the disease on which the condition is found to depend, and this must receive careful attention. The sound should be used as previously indicated under the discussion of Esophageal Stricture. Its passage has often been followed by speedy and permanent cures.

### PARALYSIS OF THE ESOPHAGUS.<sup>1</sup>

In extensive bulbar paralysis, when adjacent parts are involved, we may infer the existence of esophageal implication, though there be no

<sup>1</sup> For remarks on the treatment of this complaint the reader is referred to section on Nervous Diseases.



objective evidence to adduce in confirmation. Doubtless the esophagus rarely shares in post-diphtheritic paralysis also. Dysphagia is the leading symptom. An invaluable peculiarity belonging to diphtheritic paralysis is the fact that solids are more readily swallowed than liquids.

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## DILATATION OF THE ESOPHAGUS.

**Pathology and Etiology.**—Diffuse dilatation of the esophagus is usually secondary to stricture at or near the cardiac orifice. In accordance with the common law of compensation, the first effect of the stenosis is to engender hypertrophy of the muscular layer above it with a view of overcoming the resistance caused by the obstruction. The wall of the esophagus becomes thickened, and the tube is generally somewhat narrowed, above the seat of the stenosis; but finally, as a result of degenerative changes, the muscular coat weakens, the esophagus dilates, and food accumulates above the stricture—a condition that, once begun, progresses.

*Congenital dilatation*, in which the whole extent of the tube participates, has also been met with, though such a condition is rare indeed. It sometimes results from fatty degeneration of the muscular wall, and a predisposition to the complaint may be acquired as the result of injury or prior inflammation.

**Symptoms.**—The essential symptom is chronic *dysphagia*. When dilatation follows stenosis the patient often locates the point at which the food lodges in the esophagus. Most of the ingesta are regurgitated several hours after eating, and this process is often attended by more or less severe strangling. The *esophageal sound* comes upon the stricture, and is either gripped firmly or totally resisted; in the latter event the bulb can be moved about above this point with abnormal freedom. In the rare cases of spindle-shaped dilatation without stenosis the sound usually detects no obstacle on its way into the stomach. A *sac* is occasionally formed, however, as the result of localized bulging of the paralyzed wall, in which food may collect or the exploring sound may catch, thus leading to erroneous inferences. Dysphagia is present, though it presents peculiarities, in that the food may either pass down very slowly until it reaches the stomach, or it may find its way down for some distance and then lodge in the shallow pouch, as above described. In the latter event the food may be gulped up from time to time. If the sound can be easily introduced into the stomach, we may safely eliminate stricture as the cause of the dilatation.

The **prognosis** is good as long as sufficient food can be gotten into the stomach for the support of life.

**Treatment.**—The chief object in the treatment of this condition is to keep the patient well nourished. If sufficient food cannot be swallowed, a Symond's tube should be inserted and nourishment given through it; and when this mode of feeding is no longer feasible, the physician has to choose between gastrostomy and rectal feeding. There can be no doubt that by means of nutrient enemata nutrition may be

fairly well maintained for a considerable period of time, but not indefinitely, as these cases would seem to demand. In the hands of a competent surgeon, on the other hand, gastrostomy is often fruitful of brilliant results. Galvanism has been recommended on high authority, but I cannot speak from personal experience in its use. Local lesions, when present, must be dealt with in accordance with the rules governing the treatment of the several causal conditions.

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## ESOPHAGEAL DIVERTICULUM.

(*Pharyngocoele*.)

**Definition.**—A circumscribed sac in the wall of the esophagus.

**Pathology and Etiology.**—Two varieties are met with, which Zenker has termed *pressure* and *traction* diverticula; the latter are rare. Diverticula that occur at or near the inferior constrictor, and more particularly the larger ones, are *congenital* in origin. When *acquired* they are the result of a localized lesion in the muscular coat, through which the mucous membrane bulges like a hernia. This is owing to repeated slight pressure occasioned by the passage of food. When once such a process is started, various factors tend to continually enlarge the pouch. Chief among these are the morsels of food that find lodgement and naturally tend to augment the size of the diverticulum by dragging it downward. The sac may finally attain a diameter of not less than 4 inches (10 cm.). Its situation is nearly always on the posterior wall at the pharyngo-esophageal junction, and its form is usually saccular or pear-shaped. Most instances have been met with in males after middle life. The cause of the weakened area at which the diverticulum occurs is to be found sometimes in injury, but more frequently in an antecedent inflammation. Histologic changes are observed only in the mucous and submucous layers, these anatomic elements together forming the pouch.

*Traction* diverticula are produced by the fringe of tissues that often becomes adherent to the upper aspect of the esophagus, and from their mode of occurrence they will obviously be more or less funnel-shaped. Their dimensions are small. They are more common in children than in adults, for the reason that in the former, more frequently than in the latter, do the bronchial glands suppurate, with subsequent cicatrization. This circumstance affords an explanation of the fact that traction diverticula are usually seated on the anterior wall of the esophagus, near the bifurcation of the trachea.

**Clinical History.**—*Traction* diverticula do not, as a rule, give rise to clinical symptoms. Exceptionally, however, as the result of the mechanical irritation caused by bits of food that are retained in these funnels, ulceration may occur and be followed by perforation of their apices. In this manner the main bronchi are often perforated (causing pneumonia and pulmonary gangrene), also the pleura (causing empyema), and, more rarely, the pericardium (causing suppurative pericarditis).

*Pressure* diverticula when small cannot be recognized, owing to the

absence of signs and symptoms. When they attain considerable size, however, they are often attended with severe symptoms. The earliest clinical manifestation is difficulty in swallowing; some of the food enters the sac, and, if allowed to remain, undergoes putrefactive decomposition, causing *fetor of the breath*. From time to time, and especially on attempting to swallow, the partly or wholly filled condition of the pouch excites *nausea* and *vomiting*, associated with prolonged *strangling*; this results in the ejection of a portion of the accumulated contents. After such an attack the patient is unable, temporarily, to swallow food, and in consequence of the limited amount of food taken signs of inanition soon appear; this may finally become extreme, and is sometimes the immediate cause of death. The appearance of a *pear-shaped swelling* in the side of the neck has been observed. As the tumor enlarges it displaces the larynx and presses upon the enlarged vessels—more rarely upon the superior laryngeal nerve, giving rise to dyspnea and distressing fits of coughing.

**Diagnosis.**—A leading point in the differentiation of this affection is the enlargement of the sac after meals (not all the food passing into the stomach), and its disappearance after being emptied. Another valuable discriminating sign is the effect of compression by the hand in causing the contents (“air and sodden food”) to flow back into the mouth. In those instances in which the tumor is absent, while the symptoms point to the disease under consideration, we may demonstrate its existence by the use of the esophageal sound. If the sound passes into the sac, its descent will soon be arrested. If, however, the instrument fails to enter the mouth of the pouch, it readily glides into the stomach. An elbowed sound, bent at an obtuse angle near the tip, is especially useful in such cases. It may be inserted in different directions, so as to avoid entrance into the sac.

**Prognosis.**—The outlook is unfavorable in the absence of operative treatment, though modern surgery gives promise of curing a certain proportion of cases. Wheeler has operated successfully in one instance at least. The physician may prolong life by directing attention to the nutrition of the patient, but he cannot hope to promote a cure. If the patient cannot swallow an adequate amount of nourishment, he may be successfully fed for an indefinite period through a tube, which he himself should be allowed to pass. When sufficient food cannot be introduced by this method to maintain the powers of the patient, rectal feeding should be instituted. If excision of the diverticulum be deemed impracticable by the surgeon, then the establishment of a gastric fistula is worthy of extended trial in cases in which the above-mentioned modes of feeding have failed.

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## STRICTURE OF THE ESOPHAGUS.

**Etiology.**—A stricture of the esophagus may be due either to (*a*) Congenital narrowing (exceedingly rare); (*b*) Squamous epithelioma, usually producing an annular constriction; (*c*) Rarely to polypi protruding from the mucosa, which almost occlude the lumen of the tube;



(d) Rarely to specific inflammation, as syphilis and tuberculosis; (e) Simple stricture generally results from the ingestion of corrosive fluids, which cause extensive sloughing of the mucosa, followed by cicatricial contraction.

**Clinical History.**—The symptoms vary with the special cause and with the degree of stenosis. The first and most prominent indication of narrowing of the gullet is a very slowly increasing *dysphagia*. The patient for a long time complains of a *sense of pressure* at a certain sub-sternal point on swallowing solid food, or, more rarely, an apparently healthy person will suddenly experience painful pressure in attempting to swallow a larger quantity of food than usual. By and by even fluids cause dysphagia, and the patient observes that the time required for the food to reach the stomach is lengthened. The impediment to the act of swallowing is not due alone to mechanical stenosis, but partly to the weakness of the muscular coat, sometimes owing to its partial destruction, and in exceptional cases partly to spasmodic contraction. When due to carcinoma, difficult deglutition is, as a rule, the only symptom complained of. When occasioned by corrosive fluids or traumatism, *pain* is prominent from the onset.

Above the seat of stricture the esophagus is often *dilated* and contains accumulations of the ingesta. The latter, together with considerable mucus, are regurgitated three or four hours after meals, and we may be certain that the materials thus ejected do not come from the stomach if they are alkaline in reaction. The leading clinical features are the *gradually increasing debility* and *emaciation*, finally reaching an extreme degree.

**Diagnosis.**—However characteristic the symptoms may be, the bougie should invariably be passed before pronouncing a positive diagnosis. By this means we ascertain the degree and the seat of the stricture. To begin with, a medium-sized gum-elastic bougie (No. 16 English scale) should be employed, after warming it and lubricating with glycerin. Its use should be preceded by a cocain-spray to prevent spasm. The patient should occupy a low seat, with his head supported by an assistant from in front of the operator. The head should be only slightly thrown backward. The forefinger of the left hand should then be passed back over the tongue until it touches the epiglottis, and the bougie inserted along it with the right hand, thus avoiding the error of passing it into the naso-pharynx or the larynx. When the bougie reaches the cricoid cartilage it is sometimes gripped pretty firmly even in a healthy person—a fact that is always to be remembered. No force should be applied. The instrument may pass the constriction with a jerk, or it may not only be gripped, but distinctly arrested, when a smaller bougie should be tried. By moving the instrument upward gently we may detect sometimes several strictures lying one above another. To locate the obstacle, the distance from the teeth to the point of stricture is measured on the instrument, and the results compared with the normal measurements, which are as follows: from the teeth to the cricoid cartilage, 7 inches (17.7 cm.); to the left bronchus, 11 inches, (27.8 cm.); and to the opening into the diaphragm, 15 inches (37.9 cm.).

Auscultation of the esophagus has been practised, but the clinical indications afforded are of little practical value. The stethoscope is

placed to the left of the spine, and the patient takes a mouthful of water, when, if a stricture be present, a splashing, cooing sound will be heard at the seat of the stricture instead of the normal esophageal bruit.

**Differential Diagnosis.**—It is important to determine not only the existence of a stricture, but also the diseased underlying process, since without this knowledge rational methods of treatment cannot be instituted. First and foremost, we must exclude those affections that simulate simple and malignant stricture, in certain of which the introduction of the sound would be attended with grave dangers. *Compression of the esophagus* by enlarged or accessory thyroids, aortic aneurysms, vertebral abscess, enlarged lymphatic glands, and occasionally pericardial effusions, may produce dysphagia, and on passing the bougie resistance is offered at the seat of the external pressure. As a rule, the extent of the stenosis is moderate. If the narrowing be due to aneurysm—“(a) rhythmic movement is sometimes communicated to the free end of the sound introduced as far as the stenosis.” Careful physical examination will often reveal the presence of an aneurysm or other pressing tumor, and should never be neglected. A passage of the sound in cases of aneurysm has even caused rupture of the sac and death. (b) Spasm of the esophagus or paralysis (the latter rarely) may closely resemble true stenosis. These neurotic forms are almost exclusively met with in young hysteric females; on the other hand, malignant strictures are found almost solely in males over forty years; while in simple stricture there is usually a definite history and certain etiologic factors.

To discriminate between simple and malignant stricture is not difficult, as a rule. When a clear history of gumma, of tuberculous disease, or of injury (from corrosive liquids) is obtainable, the presence of a simple stricture may be safely inferred after eliminating the affections previously mentioned. In the absence of etiologic data pointing to the simple form, cases occurring in the male after forty years of age may be looked upon as malignant.

**Prognosis.**—In forming a prognostic opinion the chief factor to be considered is the nature of the stricture. Practically, so long as the stenosis is dilatable, the prognosis is not unfavorable provided sufficient nourishment can be taken; moreover, not a few cases of simple stricture are curable. The majority, however, come to a fatal termination finally, death resulting from exhaustion.

**Treatment.**—The chief object of the treatment is to gradually and methodically dilate the stricture in a mechanical manner. The flexible English bougie above mentioned is the best for the purpose, commencing with one of good size; conical ivory bougies, having a flexible whalebone handle, may also be used, though, being quite hard, they are apt to inflict injuries unless used cautiously. It is sometimes necessary, on account of the tightness of the stricture, to begin with a catgut sound. The method of introducing these instruments has already been given. They should be used once daily, and often can be passed successfully by the patient himself. At intervals of three or four days trials of bougies of larger size should be made. I have seen truly remarkable results from this treatment when carried forward systematically in cases due to cicatricial contraction, the patients increasing in

bodily weight and strength. In annular constrictions of a malignant type, however, the same plan of treatment is productive of temporary benefit only.

The diet deserves most careful attention. When the stenosis is so pronounced as to prohibit sufficient food being swallowed, a Symonds tube should be passed into the stomach, and through it liquid food is introduced. Concentrated forms of nourishment, as raw eggs, bovinin, and the various infants' foods, may also prove useful, and may be administered with milk. So long as an adequate amount of food (semi-solid or liquid) can be easily introduced into the stomach, the amount given should be sufficient to fully meet all the demands of perfect nutrition.

When the passage of the bougie is no longer possible relief may be secured in one of two ways: (1) rectal feeding; (2) gastrostomy, if the seat of the stricture be near the stomach, and esophagostomy if at the upper portion of the gullet. I have recently witnessed favorable results from gastrostomy in a case of simple stricture operated upon by Laplace. It is important that the patient should thoroughly masticate the food before introducing it into the stomach. Before resorting to operative procedures, however, careful trial should be made of rectal feeding, since life may be prolonged for an indefinite period by this means. Various forms of nutritious enemata and other points regarding rectal alimentation will be found in the Treatment of Gastric Ulcer.

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## VII. DISEASES OF THE STOMACH.

### METHODS OF DIAGNOSIS.

#### EXAMINATION OF THE GASTRIC FUNCTIONS.

**Secretory Function.**—When food enters the stomach the glands immediately begin to secrete their various juices, and continue to do so until the food has passed into the duodenum. During the later stages of gastric digestion the activity of the secretory function of the stomach diminishes, and to obtain accurate knowledge of any pathologic condition of the organ, examinations of the gastric contents must be made under conditions as nearly like the physiologic as possible. Reliable results cannot, therefore, be obtained from an examination of ordinary vomita, but the contents of the stomach must be procured at a definite period after a so-called test-meal (*vide infra*).

Numerous test-meals have been offered to the profession, but those that I have found most satisfactory are "the test-breakfast of Ewald and Boas" and "the test-dinner of Leube-Riegel." The former being simpler and easier of preparation than the latter, it is the oftenest used.

The *Ewald-Boas test-breakfast* consists of one or two rolls (50–70 gm.) and one cup of tea or water (300–400 c.c.). I constantly advise the use of one roll and a glass of water. About an hour after this meal has been taken the contents of the stomach are to be withdrawn, and at such a time HCl should be the only acid present.



The *Leube-Riegel test-dinner* consists of a large plate of soup (300–400 c.c.), a large piece of beefsteak (150–200 gm.), and some potatoes (about 50 gm.) or a roll—practically, a large plate of soup, a piece of meat (preferably beefsteak), and a roll of bread. The examination is to be made about three and a half to four hours after the meal.

To obtain the contents of the stomach we should use a soft, flexible-rubber tube with an end-opening, or, better still, with several additional openings on the sides, and it should be marked at a point 23.5 to 25.5 inches (58–64 cm.) from the end introduced, this helping the examiner to determine whether it has entered the fundus. The tube is moistened with water and the end carried back to the pharynx; the patient is now asked to swallow, and the tube is gently pushed down the esophagus, these acts being repeated until the tube reaches the stomach. An ordinary Politzer bag is now attached to the tube (Ewald), or a Boas aspirator (which consists of a rubber bulb having a soft-rubber tube at one extremity with clamps). If the Politzer bag be employed, it is compressed and allowed to re-expand, the contents being thus withdrawn into the bag. There are cases in which it is safer to empty the stomach by siphonage. This is readily accomplished by using a long tube and exerting gentle pressure on the abdomen to start the current.

The method I have most frequently used is that of “expression,” as follows: The patient is asked to take a deep inspiration, and then to contract his abdominal muscles as in the act of having a stool: in this way the contents are quickly expelled through the stomach-tube above described. These should be first examined microscopically to detect any residue from previous meals, such as meat and the like, and the quantity obtained should be 20 to 40 c.c. After filtering the gastric contents thus obtained they are variously tested.

Among qualitative tests the following are important:

To determine the *reaction*, ordinary litmus-paper is used; if acid, the blue turns red.

The presence of *free acids* is determined—(a) By Congo-red, a solution of which is turned blue by the addition of liquids containing free acids. The use of Congo-paper (prepared by dipping ordinary filter-paper in Congo-red solution) is the easiest method.

(b) *Tropeolin O. O.*—Alcoholic solutions of tropeolin are turned by the addition of liquids containing free acids to a brownish-red, deep red, or deep mahogany-brown, according to the amount of acids present. Tropeolin-paper (filter-paper immersed for some time in an alcoholic solution) may be used, but must not be kept too long.

*Free HCl.*—*Günzburg's test*—phloroglucin gr. xxx (2.0), vanillin gr. xv (1.0), absolute alcohol 5j (30 c.c.). To two or three drops of this reagent add an equal number of the gastric filtrate in a porcelain dish, and slowly evaporate to dryness over a flame; and if free HCl is present, a rose-red tint appears along the edges. Blowing at the edge will hasten the reaction. The great delicacy of this test is conclusively shown by its availability when HCl is present in the proportion of 1 to 20,000. There are no recognized interfering conditions.

*Boas' Resorcin Test.*—Resublimed resorcin 5 parts, white sugar 3 parts, and diluted alcohol 100 parts. The method of procedure is the same as in Günzburg's test, and a purple-red color appears. More

caution is required in evaporating, but this method will also detect the presence of free HCl in the proportion of about 1:20,000.

*Lactic Acid.*—*Uffelmann's test.* The reagent should always be freshly made, as follows: To 10 or 15 c.c. of a 2 per cent. aqueous solution of carbolic acid add 1 or 2 drops of neutral ferric chlorid, when an amethyst-blue color will appear. To 1 or 2 c.c. of the mixture add a few drops of the filtrate, and if lactic acid is present it changes to a canary-yellow color. Certain substances that are often found in the stomach, as alcohol, sugar, and various salts, especially the phosphates, may give a coloration simulating that of lactic acid. These sources of error may be overcome by shaking 5 or 10 c.c. of the filtrate with double the quantity of ether, and, after allowing the ether to separate and pouring it off, more is added, the whole shaken, and the washing is repeated. The ether is then decanted and evaporated almost to dryness in a water-bath. To the residue about 1 c.c. of water is added, and to this an equal quantity of the Uffelmann reagent from a pipette; if a canary-yellow now appears, positive proof of the presence of lactic acid is afforded. Boas and others have experimentally shown to their own satisfaction that the presence of lactic acid in the gastric contents during the first stage of digestion (formerly believed to be physiologic) has pathologic significance. Boas also found that ordinary bread contains lactic acid, and hence he has abandoned the usual test-meal, so far as the determination of this acid is concerned, and adopted a thin gruel made by adding to a quart of water flavored with salt half an ounce of oatmeal-flour. Boas states that no lactic acid is present in the filtrate several hours after this test-meal, except in cases of carcinoma of the stomach. The use of this test-meal for usual clinical purposes is now generally held to be superfluous. Lactic acid in the stomach-contents occurs with fermentation-stagnation from either obstruction or deficient motility.

A more reliable test for lactic acid than the foregoing is that of Boas, as follows: Digest the filtrate several times with ether to remove the fatty acids; add a few drops of phosphoric acid and boil. Transfer the mixture to a distillate flask; add  $\text{H}_2\text{SO}_4$  and  $\text{MgO}_2$ ; heat, and lactic acid will be distilled over. This can be conducted into a strongly alkaline solution of iodine and potassium iodid. The presence of lactic acid is then shown by the production of iodoform, which can be recognized by its odor and by the precipitate that is formed.

*Fatty or Volatile Acids.*—Heat to boiling a few c.c. of the filtrate in a test-tube, over the mouth of which place a strip of moistened blue litmus-paper; the presence of fatty acids will change the paper to red.

*Acetic Acid.*—In large quantities this acid is detected by its odor, and in smaller quantities its presence is determined by neutralizing with sodium carbonate the watery residue of the ethereal extract, and adding neutral ferric chlorid, when a blood-red color will be struck. Quantitative estimation of certain constituents is desirable.

*Total Acidity.*—To 10 c.c. of the filtrate add 1 or 2 drops of a 1 per cent. alcoholic solution of phenolphthalein, and as many cubic centimeters of a decinormal solution of sodium hydrate are added slowly from a buret until the reddish color that appears fails to disappear on shaking. The number of cubic centimeters of the decinormal solution normally required

ranges from 4 to 6; hence, if these be multiplied by 10, we have 40 to 60 as the percentage of acidity. Under pathologic conditions these numbers may be either higher or lower. This total represents both free and combined acids. If no organic acids be present, the above figures will represent the percentage of HCl. The latter is also reckoned thus: If it required 5 c.c. of the decinormal solution of sodium hydrate to be added to 10 c.c. of the filtrate to get the red color (alkalinity) with the phenolphthalein, we say the acidity is 50, and multiplied by  $0.003,646 = 0.1823$  per cent. of hydrochloric acid. It should be stated that the normal range of percentage is from 0.14 to 0.24.

*Estimation of Free HCl.*—*Mintz's method:* To 10 c.c. of the filtrate add a decinormal solution of sodium hydrate from a buret until no reaction is given with Günzburg's reagent. The number of c.c. of the decinormal solution used, multiplied by 10 and then by 0.003,646, gives the percentage of free hydrochloric acid.

*Estimation of Lactic Acid.*—If the volatile acids are present, they should be removed by boiling. Take the total acidity of 10 c.c. of the filtrate; then to a second 10 c.c. add 25 to 30 c.c. of ether; shake well, allow the ether and filtrate to separate, remove the ether, and again add 25 to 30 c.c. of ether; shake, and repeat the process. Next obtain the acidity of the watery solution, and the difference between this and the total acidity, multiplied by  $10 \times 0.09$ , will give approximately the amount of lactic acid.

In the *gastric digestion* of the *albuminoids* (proteolysis) the proteids are converted into peptone. Although commenced in the stomach, this function is dependent in greater part upon the action of the pancreatic ferment in the small intestines. Among the substances earliest engendered by this process are the *albumoses* (propeptone), whose separation may be thus effected: Add a small quantity of a saturated solution of sodium chlorid to an equal amount of gastric filtrate, and if it becomes cloudy propeptone is present, the degree of the cloudiness indicating the amount present. If the mixture does not become turbid, add a few drops of acetic acid, when it will become so in the presence of this substance, however slight the quantity. If heated, the solution becomes clear, and if allowed to cool, the propeptone precipitates and may be obtained by filtration.

In a later stage of the process of albumin-digestion peptone is produced and its detection is easy. To a small quantity of the filtrate (the propeptone having been removed) add enough sodium or potassium hydrate to render the solution alkaline; then add a few drops of a 1 per cent. solution of cupric sulphate, and, if peptone be present, a purplish color is presented.

*The Test for Pepsin.*—In a test-tube containing 5 c.c. of filtrate add a small piece of egg-albumin, and keep at a temperature of about  $100^{\circ}$  F.; if present, the albumin disappears in from two to six hours. If hydrochloric acid is absent from the filtrate, it is necessary to add a few drops of the dilute acid. It should be pointed out that laboratory attempts to estimate the rate of albumin-digestion are unreliable.

*Rennet Ferment.*—To 5 or 10 c.c. of raw milk add a few drops of the gastric filtrate, and keep it at a temperature of about  $100^{\circ}$  F.; if rennet



is present, coagulation into a single cake occurs in from a few minutes to an hour or more.

*Rennet Zymogen* (which is converted into *rennet ferment* in the presence of an acid).—To 5 c.c. of gastric filtrate add enough sodium carbonate or sodium hydrate to make it slightly alkaline; then add calcium chlorid (1–2 c.c. of a 2 per cent. solution); then mix with an equal quantity of milk, and, if zymogen is present, coagulation occurs as in the case of rennet ferment. Both rennet ferment and rennet zymogen may be assumed to be present when HCl has previously been found.

*Starchy Derivatives*.—To a small quantity of gastric filtrate add 1 or 2 drops of Lugol's solution; the presence of dextrin gives a blue reaction—erythrodextrin purple, achroödextrin, grape-sugar, and maltose (intermediate substances)—showing a yellowish color. If there is a mixture of these starchy derivatives, as when the digestion of starches proceeds naturally, the first few drops of Lugol's solution may produce no color-reaction, or it may be taken up by the dextrose or maltose, while the addition of more of Lugol's solution will give a purple (if erythrodextrin be present) or a blue color, due to starch.

Indeed, if a minute quantity of the solution strikes a blue or purple tinge, conversion of starch into maltose has been abnormally tardy. I believe this is oftenest due to hyperacidity, though it may also more rarely be due to a defective ptyaline-supply.<sup>1</sup>

**The Tests for the Motor Function.**—More important than the secretory is the motor function of the stomach. There are three recognized tests:

The oldest method is that of *Leube*. It consists in washing out the stomach from six to seven hours after a large meal, preferably consisting of beef-soup (13 oz.), beefsteak ( $6\frac{1}{2}$  oz.), bread ( $1\frac{1}{2}$  oz.), and water ( $6\frac{1}{2}$  oz.), or from two to two and a half hours after Ewald's test-breakfast. Normally, the stomach should be empty within these periods of time, so that if a residue remains it denotes a lack in the motor force.

*Salol Test of Ewald and Sievers*.—Salol being composed of phenol and salicylic acid, it is not acted upon in an acid medium; therefore, when introduced into the stomach it remains a stable compound, and is only broken up in the intestine by the action of the pancreatic juice. The salicylic acid is absorbed into the blood and eliminated through the urine, in which it can be detected by adding a few drops of neutral ferric chlorid, a violet color appearing. The patient is given 15 grains (1 gm.) of salol in two thin gelatin capsules; the bladder is emptied, and the patient told to urinate every half hour for two hours. Normally, it requires from three-fourths to an hour for the salicyluric acid to appear in the urine, but when the motor function of the stomach is much impaired it may require two or more hours. In order to detect the earliest traces of the eliminated salicyluric acid, Ewald and Einhorn have suggested moistening a piece of filter-paper with the urine, and then allowing a drop of neutral ferric chlorid solution to come in contact with it, the edges of the drop showing a violet color in the presence of the slightest traces. The varying reaction of the intestinal contents

<sup>1</sup> The tests for the estimation of the combined acids, of some of the fatty acids, and of many of the products of proteolysis are complicated and unnecessary in an ordinary clinical examination.

renders the salol test very unreliable. Sometimes in healthy individuals the decomposition of the salol is retarded, and to overcome this objection Huber has suggested the determination of the precise time when the salicyluric acid no longer appears in the urine. He found that normally it required from twenty-four to thirty hours. If, then, the reaction outlasts this period, it shows peristalsis or the motor function to be unduly tardy. Leube's test is much more reliable.

*Klemperer's Oil-test.*—The stomach is thoroughly washed, and  $3\frac{1}{2}$  ounces (100 c.c.) of olive oil are poured into it through the tube. Two hours later the remaining oil is withdrawn by aspiration. As the stomach-wall does not absorb oil, the difference between the original amount and that withdrawn shows the condition of the motility. Klemperer states that at this time the residue should not exceed 20 to 40 c.c. This test is also unreliable.

**To Test the Absorptive Power.**—The method described by Penzoldt has been almost universally adopted: A capsule containing grains  $1\frac{1}{2}$  (0.1), of potassium iodid is given to the patient, care being taken that the capsule is first carefully wiped. The iodid is absorbed from the stomach and appears in the saliva, which is to be examined for the presence of iodine. For this purpose strips of starch-paper (filter-paper moistened in a solution of starch and dried) are used; they are moistened with the saliva of the patient, and the moistened areas treated with a drop of fuming nitric acid. As soon as the iodine enters the saliva, the characteristic reaction for starch is struck—a blue color. Normally, this reaction occurs in from ten to fifteen minutes; under abnormal conditions it may be delayed for half an hour or more. Rarely it fails to occur. This test cannot be strongly depended upon.

#### PHYSICAL OR EXTERNAL EXAMINATION.

This implies the well-known physical signs—inspection, palpation, percussion, succussion or splashing, and auscultation.

**Inspection.**—(a) *General.*—This may give an idea of the nature of the illness as well as its severity by noting whether the patient appears to belong to a neurotic group, the general health often being good, or whether the patient is emaciated, or has with the latter the cachexia of a malignant growth. In diseases of the stomach attention should be directed to the mouth, and especially to the teeth, because the latter are often of causal importance in many gastric ailments. Dental affections often prevent the possibility of curing the various chronic diseases of the stomach.

(b) *Local Inspection.*—In patients with thin and relaxed abdominal walls the contour of the stomach can be plainly noted; especially is this the case in very large, dilated stomachs or in those that have been displaced. The examiner is greatly aided by inflating the stomach with air or gas. The former is to be preferred, for the reason that the supply is easily regulated; he is enabled to watch the different steps of the distention, and after the examination is completed the air is allowed to escape through the tube. For this purpose an ordinary stomach-tube is most convenient, and its passage is to be effected in the same way as in removing the gastric contents. A double bulb-attachment is connected

with the external end of the tube, by means of which air is readily forced into the stomach (*Runeberg's method*).

*Frerichs' method* is sometimes used. It consists in administering 3j (4.0) of tartaric acid, dissolved in half a glassful of water, and immediately afterward 3j (4.0) of sodium bicarbonate, dissolved in the same amount of water. Effervescence now occurs, with a progressive visible distention of the organ. The chief objection to this method is the fact that either too much or too little distention is obtained.

The inflated stomach presents a circumscribed protuberance, usually in the epigastric, and also in the umbilical region if the organ is dislocated or dilated. The air may find its way into the intestine, producing a visible change in the contour of the abdomen. Tumors and other abdominal enlargements may also be recognized, and an idea obtained as to which organ is involved, after making due allowances for displacement, as in gastropnoia and pyloric carcinoma. Exaggerated peristaltic waves may also be noticeable in the upper portion of the abdomen, usually when associated with the stomach, and in the lower portion if it is in the small intestine. Peristalsis is increased from various causes—inflation of the stomach, external tapping, neuroses, pyloric obstruction, and the like.

The value of the gastroscope in inspecting the interior of the stomach is, I think, doubtful. Gastro-diaphany (illumination of the stomach) is sometimes useful in showing the fundus extending to a lower level (at the navel) than is indicated by percussion. The Röntgen rays (skiagraphy) already enable us to detect non-penetrable foreign bodies in the stomach, and bid fair to distinguish aneurysms, gall-stones, and the like in the interior of the body.

**Palpation.**—This elicits at times more trustworthy information than inspection. The patient should be in the recumbent position, the lower limbs partially flexed on the abdomen and the head low. The examiner should stand at the right side of the patient and use the right hand, which should be warm. With the palmar surface down gentle pressure should be made with the fingers and the ulnar side of the hand. If the abdominal wall is tense, it is best to distract the attention of the patient from the examination by talking to him. In this manner we can corroborate the inspection as to the size, shape, and position of the stomach, and can detect morbid growths and determine their consistency and movability. Caution must be exercised to differentiate between normal and abnormal conditions, and opportunities should not be neglected by the physician to equip himself in delicacy of palpation by practice upon a normal abdomen.

*Deep* palpation elicits the degree of sensitiveness, tenderness, or pain, whether circumscribed as in ulcer or diffuse as in generalized inflammatory states (enterocolitis, peritonitis). In some instances relief from pain may be noted on pressure with the broad hand in neuroses. Variations in the degree of tension and of resistance are found and prove valuable aids.

With Boas's algometer we are enabled to detect the amount of pressure necessary to be exerted over a given area to cause pain, by reading the number of kilograms from a scale. In some instances this is a serviceable instrument, but in ulcer, when palpation must be done



with the greatest gentleness, it would not be a very safe procedure as compared with the soft hand—nature's own instrument.

**Percussion.**—The patient is placed in the recumbent position; the examiner uses his fingers and endeavors to discriminate the slightest differences in the note, and percusses lightly. If the stomach is empty or partially filled with gas, it gives a lower tympanitic sound than the colon, which is also often filled with gas. To ascertain the size and position of the stomach by percussion the process should begin at the symphysis pubis and follow the median line upward. The upper border of the stomach is at the ensiform cartilage, the lower about two fingers' breadth (3 cm.) above the umbilicus; hence, if the latter is below the umbilicus and the upper border in the normal position, it denotes an enlarged stomach. If the upper margin is some distance below the ensiform, displacement of the organ is indicated.

It is well to trace the limits of resonance of the stomach and of any areas of dulness met with, so that their size and position may be graphically represented. The differences in the percussion-note over the stomach and colon may be greatly exaggerated by inflating the former. Runeberg's method is to be preferred. By employing light percussion the limits of the stomach can now be easily and accurately defined, unless the transverse colon be at the same time greatly distended with gas. In such instances Dehio's modification of Piorry's method is to be resorted to. It consists in giving about 1 liter (1 quart) of water in fractional doses while the patient is standing; one-quarter of the liter is swallowed and percussion practised, when a dull note will be obtained over the most dependent portion of the stomach. A second quantity of equal amount is given and a re-examination made, and so on, the object being to ascertain to what point the lower border sinks on the addition of more fluid. Boas holds that this method tests effectively the tone of the stomach, and that a *marked* descent of the lower border after each addition of water is indubitable evidence that there exists weakness or atony of its walls. If a neoplasm originates posterior to the stomach or colon, inflation of the latter may cause the previous circumscribed dulness to disappear.

By striking the abdomen in the epigastric region splashing-sounds may be produced. This sign is of diagnostic value in dilatation of the stomach, though its absence does not contradict the presence of the dilatation. Again, if the splashing-sound is obtained in a fasting stomach, it may give a clue to some abnormal condition. In many instances, however, the stomach may contain large quantities of fluid and no splashing-sound be obtained. Caution should be exercised lest the splashing-sound sometimes produced in the transverse colon be mistaken for that originating in the stomach; in the former the sound is usually associated with diarrhea, while in the latter constipation usually obtains.

**Auscultation.**—Various sounds are heard, none of which are pathognomonic of any diseased condition.

*Succussion-sounds* are produced by shaking the patient, and, if the stomach is dilated and contains fluid, the sounds may be audible some distance from the patient. The patient can sometimes engender similar sounds by voluntary contraction of the abdominal muscles. Various murmurs are heard in the act of deglutition—one when the food passes

from the pharynx into the esophagus; Ewald has described two, both heard at the cardiac extremity of the stomach; the first is a hissing murmur, the second a splashing or sprinkling. The heart-sounds are heard over an inflated stomach, and have a clear, metallic quality.

*Sizzling sounds* are audible when fermentation occurs; also after giving a Seidlitz powder.

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## MALPOSITION OF THE STOMACH.

THE stomach may occupy a truly vertical position in consequence of the persistence of the normal infantile condition or of improper clothing, as long-continued pressure from corsets and the like. Unless an angular condition of the duodenum, causing obstruction to the outflow of the gastric contents, followed by dilatation of the stomach, be engendered, the malposition is of little or no clinical significance. Transposition of the stomach, with the organ occupying the right hypochondrium, is rarely met with in association with transposition of other viscera.

### GASTROPTOSIS.

**Definition.**—Downward displacement of the stomach. The lesser curvature of the organ lies about midway between the ensiform cartilage and the umbilicus, and the greater curvature near the symphysis pubis.

**Etiology.**—So far as our present knowledge extends, the conditions and circumstances contributing mostly to the origin and development of gastroptosis are—(a) Age and sex. Meinert of Dresden found among girls of fourteen years gastroptosis in 80 per cent., and among the women who presented themselves at his private clinic in 90 per cent. According to my own observation, gastroptosis is not as frequent among American girls and women as among the Germans. “Dislocation occurs in about 5 per cent. of the male population of Dresden.” (b) Improper clothing, particularly tight lacing. (c) Dislocation of the right kidney. This operates potently, and prolapse of other abdominal organs may occasionally constitute the chief point of departure. (d) Repeated pregnancies, inducing a relaxed state of the abdominal wall. (e) Muscular strain and local injury, by diminishing the tonicity of the gastro-hepatic omentum. (f) Abnormalities of the chest-formation (kyphosis); great meteorism, and enlargement of the abdominal organs, especially of the spleen and liver. Certain chronic diseases may be active—*e. g.* chlorosis, tuberculosis.

**Symptoms.**—Malposition of the stomach may exist without symptoms, but in most instances it produces functional disturbances of clinical importance. The latter are due, first, to the difficulty that the stomach experiences in emptying its contents. Soon functional disorders arise in consequence of gastric atony, and later there is apt to be a greatly diminished gastric secretion, associated with a nervous dyspeptic condition. Especially to be emphasized is the fact that the stomach may be of natural or of diminished size (as the primary result of the compression of the corsets—Fleiner), or it may be dilated—a not

uncommon event that often colors the clinical picture in a peculiar manner. *Constipation* due to defective peristalsis, and *colicky pains* due to spasm of the intestinal muscles, are important features.

*Physical examination* of the inflated stomach<sup>1</sup> permits the accurate demonstration of gastrop-tosis. The percussion-note now indicates the position of the organ. It is to be borne in mind that the cardiac end remains fixed at the twelfth dorsal vertebra, while the pylorus moves downward and to the left: this will explain why the epigastrium is free of gastric tympany. *Succussion splashing-sounds* may be heard if atony, with retained gastric contents, obtains. The differentiation of gastrop-tosis from dilatation of the stomach is also accomplished by the method of inflation, since this makes plain the course and position of the lesser curvature and of the pylorus.

The **prognosis** is not bad, being much the same as in nervous dys-pepsia; it is modified in some cases by the presence of special causal agencies, and in others by the occurrence of certain complications, as dilatation of the stomach.

The **treatment** has relation to the removal of all causes that favor the condition and to the associated functional disturbances. Nervines and nutrients are especially to be employed.

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## DILATATION OF THE STOMACH.

THE condition is to be subdivided, clinically, into acute and chronic forms. The normal capacity of the stomach varies within rather wide limits, though the maximum normal capacity, according to Ewald, does not exceed 1600 c.c. (1.5 quarts); enlargements above this capacity may then be said to fall under the heading of dilatation.

**Pathology and Etiology.**—The chief factor in the production of chronic dilatation is **pyloric stenosis**. This is usually due (*a*) to such diseases of the stomach as carcinoma, ulcer (occasionally), hypertrophic thickening of the pylorus, or the thickening and induration consequent on the action of corrosive poisons; (*b*) to the external compression arising from carcinoma of the liver, pancreas, or gall-bladder, from the omental lymph-glands, and not infrequently from a displaced right kidney, or from large gall-stones; (*c*) congenital pyloric stenosis and adhesions about the pylorus may also act as causes of dilatation.

Obviously, in all such instances increased force is necessary to propel the food from the stomach into the duodenum, thus leading gradually to a hypertrophy of the muscular fibers; this is noted in an especial degree in the immediate vicinity of the pylorus. So long as this hypertrophied state of the muscular layer compensates for the obstructive lesion no pathologic dilatation can occur. Just as soon, however, as the muscles prove to be inadequate on account of secondary degenerative changes, accumulation of the undigested food in the stomach ensues. This tendency for the contents of the stomach to accumulate is

<sup>1</sup> Inflation may be accomplished by the use of effervescent mixtures or by the introduction of atmospheric air (*vide ante*).



very much augmented by the increasing weakness of the muscle on the one hand and the increasing degree of stenosis on the other. A further step in the anatomic alteration is the development of a chronic gastric catarrh in consequence of the chemical and mechanical effect of the undigested food, the latter inevitably undergoing fermentative and putrefactive changes from prolonged retention. The degree of dilatation is, to some extent, augmented by the generation of excessive amounts of gases under these abnormal conditions, as well as by the great weight of the accumulated gastric contents. When produced in this manner the stomach attains enormous dimensions, and one instance has been recorded in which it was capable of containing 90 pounds of fluid (Loomis). Dilatation is usually general, though there may be mere diverticula corresponding to the seats of ulcers or to erosions of the walls.

Dilatation may also occur *independently of pyloric stenosis*, though this class of cases is not so large as the preceding, nor does the condition attain so pronounced a degree. In this variety of dilatation there is atony of the muscular coats, due to various and dissimilar causes: (a) repeated over-strain of the muscular layer, due to over-filling of the organ with food and drink, is a comparatively frequent cause, and one met with in diabetics and in those who habitually drink large quantities of beer; (b) chronic gastric catarrh frequently weakens the muscle, and more especially when associated with an over-indulgence in food and drink; (c) fatty and other forms of degeneration or nutritional disturbances associated with certain constitutional diseases (particularly carcinoma, anemia, and tuberculosis); (d) congenital weakness of the muscular coat; (e) impaired innervation, leading to imperfect peristalsis and consequent dilatation; (f) omental hernias (Bamberger) that drag down the stomach; (g) fibrous bands, by binding the stomach to other organs, will occasionally cause slight dilatation.

*Acute dilatation* has for its chief causes—(a) specific fevers, producing parenchymatous degeneration of the muscular coats; (b) the acute paralytic distention of Fagge, who ascribed the condition primarily to chronic catarrhal inflammation; (c) the drinking of large quantities of effervescing liquids; (d) Boas and Rosenheim have recently reported cases of acute dilatation following shock.

**Clinical History.**—Since the diseases causing dilatation are numerous and diverse, the clinical history presents great variations. Associated with the symptoms of dilatation are usually those of the causal affections, and the latter sometimes overshadow the former. Among the earlier symptoms, *increased hunger and thirst* are frequently observed, partly due most probably to the general condition of inanition. The thirst is also due, according to Von Weinig, to the fact that the stomach does not readily absorb water, and the pyloric obstruction prevents the passage of water into the intestines. *Vomiting* occurs at intervals of several days, the matter ejected amounting to from 1 to 3 gallons (4–12 liters). The clinical characters of the *vomit* are strikingly peculiar. Occasionally the vomiting occurs more or less regularly some hours after feeding. The ejecta often contain remnants of previous meals, are, as a rule, excessively acid, emitting a sour odor, and on microscopic examination they show bacteria, sarcinae, and torulae in great numbers. The vomitus undergoes fermentative changes very rapidly, is ill-smelling, the odors being mainly due to sulphuretted and phosphuretted hydrogen. It con-

sists of acetic, butyric, and lactic acids and partially decomposed food, and on standing separates into three layers—an upper layer of brownish froth, a middle one of grayish-brown fluid, and a lower one composed of remnants of food. The acid contents of the stomach are not infrequently regurgitated, causing *pyrosis*. Eructations of foul gases are also common, and certain *general symptoms* almost invariably ensue. Progressive emaciation naturally follows, sometimes becoming extreme. A characteristic symptom is *muscular cramp* affecting the calves of the legs and sometimes spreading to the flexors of the arms and the abdominal muscles; Kussmaul attributes this to an abnormal dryness of the muscular tissue. It is similar to the cramps in cholera. Owing to the fact that but a small amount of liquid reaches the intestines, and also to the impaired absorption from the stomach, there are *constipation* and *scanty urine*, the latter usually being alkaline in reaction. *Loss of consciousness* has been met with, and *tetany* has also rarely been observed. In two instances in which lavage was employed in the treatment of dilatation tetany followed, lasting three-quarters of an hour; death followed about seven hours after the beginning of the attack in both instances. A striking instance is reported by J. T. Whitcomb in which nearly all the muscles of the body, including those of the esophagus, appeared to be in a tetanic condition. Dreschfield calls attention to *dilatation of the epigastric veins* in both inguinal regions as an evidence of dilatation of the stomach; he has observed it in more than 60 cases, and refers to compression of tributaries of the portal vein by the dilated and displaced stomach as the supposed cause of the condition.

**Physical Signs.**—*Inspection* reveals a rounded prominence just above the umbilicus in the supine and just below the umbilicus in the standing posture. In the epigastric region there is sometimes a noticeable depression. Obviously, then, the abdomen is asymmetric in appearance. The outlines of the stomach may be made distinct by the patient taking an effervescing draught, and may sometimes be readily seen, particularly the greater curvature, “passing obliquely from the tip of the tenth rib on the left side toward the pubes, and then curving upward to the right costal margin” (Osler). Sometimes peristalsis is visible through the abdominal walls, and rarely the peristaltic waves are seen passing from right to left. These movements may be excited mechanically by various manipulations. *Palpation*.—The increased resistance of the walls of the stomach and their peculiar elasticity aid us in mapping out the contour of the stomach with more precision by palpation than by inspection alone. The movements of the organ can be plainly felt, frequently leading up to a pyloric mass. A sign of considerable diagnostic importance is the loud splashing sound obtained by tapping the region of the stomach with the finger-tips of both hands alternately, though this should not be mistaken for a similar sound that may be obtained when the colon contains fluid. The patient may produce, and maintain for a considerable period, similar splashing sounds by breathing rapidly and forcing down the diaphragm at the same time. His own bodily movements may provoke them. *Percussion* furnishes subsidiary evidence as compared with palpation. The examiner should first percuss the empty, and then the filled stomach, if he would obtain reliable aid from this sign. When empty, an increased area of tympanitic resonance will be obtained, extending from above downward to a point several inches

below the umbilicus. If now water amounting to 1 quart (1 liter) be introduced into the organ, and, in consequence, a line of dulness at or below the navel be noted where tympanitic resonance had been found, we have good evidence of the existence of dilatation. The posture of the patient should next be changed, when it will be found that the line of dulness has also altered. Frerichs' plan of expanding the stomach with carbon dioxid, and also Runberg's method (*vide* Physical Examination), may be employed as aids to mapping out the limits of the organ. *Auscultation* reveals little that is of diagnostic value. The transmitted sounds heard over the stomach have a metallic ring. I have confirmed the observation by Franck and others, who claimed to have heard peculiar gurgling sounds produced by the heart's action and systolic in rhythm. Fluids swallowed by the patient may be heard dropping into the dilated stomach, and succussion-sounds may be elicited by shaking his body. *Measurements* made by introducing a probang into the stomach until it reaches the greater curvature are valuable only when the degree of dilatation is considerable, on account of the obvious chances of error. In health the instrument passes about 60 cm. (24 inches), reaching a point more or less nearly on a level with the umbilicus, while under conditions of extreme dilatation it may be introduced 70 cm. (28 inches).

**Diagnosis.**—The diagnosis embraces, first and foremost, the recognition of the special causes. The unmistakable clinical manifestations are the characters of the vomitus and the peculiar manner of recurrence of the vomiting. These, together with the physical signs and a knowledge of the causal condition in the individual case, are adequate for a positive diagnosis.

**Differential Diagnosis.**—The condition is most apt to be confounded with *ascites* or *over-distention* of the bowel, and in the female with *ovarian cyst*. In *dilatation of the intestines* the gastric symptoms of dilatation of the stomach are wanting; moreover, the physical signs are dissimilar. The splashing sounds on manipulation, the line of dulness below the umbilicus after filling the stomach, and other signs so significant of gastric dilatation are absent in over-distention of the intestines. In addition, we should make trial of the salol test, though this is now considered of little value (*vide* Chemical Examination). In dilatation of the stomach salicyluric acid appears in the urine two or three hours after salol has been taken, while in health as early as from one-half to one hour. From dilatation of the stomach we may discriminate *ascites* by the history and by the characteristic gastric symptoms belonging to the former affection. In dilatation the abdomen is asymmetric, the projecting prominence being in the vicinity of or just below the umbilicus. In ascites the lower portion of the abdomen is chiefly distended, and on assuming the recumbent posture the abdominal area becomes broadened and flattened. On palpation fluctuation may be elicited in the hypogastric and iliac regions. *Megalogastria*, or simple "big stomach," is distinguished by its absence of symptoms, and especially by the fact that the food is passed into the intestines as quickly as in health. *Gastroptosis* is easily distinguished from gastric dilatation (*vide* article on Gastroptosis).

**Acute Gastric Dilatation.**—Acute dilatation of the stomach has a sudden onset, and gives rise to all of the above-mentioned physical signs.



In this type of the disease, however, vomiting is more frequent and severe than in the chronic form. Cyanosis is a common symptom, and pain often a prominent one. The patient frequently passes into a condition of collapse that may prove speedily fatal. Acute dilatation may arise in the course of chronic cases, or may be primary. When it occurs independently of the chronic variety, it is often recovered from in the course of two or three days.

**Prognosis.**—The prognosis in the *acute form* is uncertain, though the majority of cases recover; the condition may, however, tend to merge into the chronic form.

*Chronic dilatation* offers a bad prognosis, most instances being utterly incurable. Obviously, it depends greatly upon the causal conditions. A resort to surgical interference sometimes gives promise of a more favorable subsequent course in cases of cicatricial stenosis. Cases of dilatation that are not secondary to pyloric obstruction, however, give a more favorable prognosis on the whole.

**Treatment.**—One of the chief aims of the physician should be to lessen the labor of the muscular coat and to prevent the continual necessity of passing the contents of the stomach into the intestines. This is to be accomplished by careful attention to the character and amount of food taken and by frequent cleansing of the stomach. It is necessary to thoroughly empty the organ by lavage, this being repeated daily. A thorough and safe manner of washing out the stomach is by means of the soft Nélaton catheter, its introduction being unattended by injurious local effects. Perhaps the best way in which to thoroughly empty the stomach is by the use of the stomach-tube, as will be detailed under Chronic Gastritis. Recently this has been replaced by the siphon apparatus as a simpler and more convenient mechanism than the former, and one not so likely to be attended with harmful effects, though perhaps less efficacious. The long course of these conditions renders it desirable that the patient should, whenever possible, be taught to wash out his own stomach. On account of the fermentative and putrefactive changes going on in the ingesta it is necessary to use weak antiseptic solutions for this purpose, suitable ones being a 3 per cent. solution of boracic acid or a 1 per cent. solution of salicylic acid. Subsequently warm water alone may be employed. The *diet* should be composed chiefly of fluids, given in small quantities and at stated intervals. If the pyloric obstruction be not too far advanced, tender meats, eggs, and other easily digested albuminous articles of food may be allowed in moderate quantities. Since gastric digestion and absorption are very often markedly impaired, it is well also to include those substances that are readily digested and assimilated after leaving the stomach, though the latter must be given in a fluid state. Too much care and attention cannot be bestowed upon the question of the adaptation of the diet to the condition of the patient. In no other manner can we bring such marked relief from disagreeable gastric symptoms as by a suitable dietary, and in no other manner can the general nutrition of the patient be so markedly improved. The weakened condition of the muscle-walls is due to over-strain and to degenerative processes; hence, after having removed as much of the labor thrown upon it as possible, we should attempt to overcome its parietic state by the employ-

ment of such agents as strychnin and electricity. For the associated catarrhal state the remedies recommended under Chronic Gastric Catarrh may be employed.

Since some of the more annoying symptoms and remote evil consequences are directly attributable to the fact that too small a proportion of the gastric contents finds its way into the intestines, we should compensate for this deficiency of intestinal fluid by rectal injections of a weak solution (gr. v to ʒj—0.324 to 32.0) of sodium chlorid, not less than one pint of this solution being injected twice daily. In addition, nutrient enemata should be employed when, despite proper regulation of the dietary, loss of flesh and strength continue. In consequence of the marked anemia and extreme emaciation frequently present in this affection tonics are indicated, and more particularly iron, which may be administered hypodermically in the form of the albuminate. Finally, it may be necessary to resort to surgical measures.

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## INFLAMMATORY DISEASES OF THE STOMACH.

### ACUTE CATARRHAL GASTRITIS.

(*Acute Gastric Catarrh.*)

**Definition.**—An acute catarrhal inflammation of the mucous membrane of the stomach, attended with more or less severe local and constitutional symptoms.

**Pathology.**—The postmortem evidences of an acute inflammation of the gastric mucosa are distinctive only of the graver forms, since the latter alone usually terminate fatally. Observations upon cases of gastric fistula, however, have shown that in the milder grades of acute gastric catarrh the morbid appearances are similar to those characteristic of acute catarrhal inflammations of the portions of mucous membrane normally exposed to view. Thus, at first there are small irregular patches of redness, slight swelling, dryness, and ecchymosis. Later, serum effused from the congested vessels, and mixed with an increased quantity of mucus, escaped leukocytes, and desquamated epithelium, is present. Hemorrhagic erosions may be seen; the mucous membrane is now thickly swollen, softened, and covered with a tenacious mucus, which, from an increase in the number of leukocytes in a more intense inflammation, may pass into a purulent exudate. Infiltration and swelling of the solitary lymph-follicles are frequent; these sometimes form minute abscesses that burst and result in follicular ulcers. The gastric tubules may be filled with a granular debris of epithelial cells that have undergone albuminous infiltration (cloudy swelling) and fatty degeneration.

**Etiology.**—The *predisposing causes* of acute gastric catarrh embrace those various impairments of the system in which the normal functional activity of the stomach is altered or diminished. These are seen as the result of (*a*) improper hygienic surroundings; (*b*) malnutrition; (*c*) the various anemias; (*d*) in gouty and rheumatic subjects; (*e*) in the

tuberculous, cancerous, and malarial dyscrasæ; (*f*) associated with chronic passive hyperemia of the stomach due to emphysema of the lungs, cirrhosis of the liver, and renal and cardiac diseases; (*g*) in sickly and delicate children, in convalescents from acute diseases, and in enervated chronic invalids. (*h*) Persons having chronic gastric catarrh are predisposed to superadded attacks of the acute disorder.

The *direct causes* are mainly (1) dietetic. These include the ingestion of too much indigestible food; food that is too hot or too cold; very sour and highly seasoned articles; the too free use of condiments; and especially the eating of decomposed canned goods and tainted meats. In cases due to the latter the fermentative and putrefactive agents (acetic, lactic, and butyric acids, and the ptomaines) are the immediate causes of the catarrhal inflammation and tend to produce the constitutional disturbances, sometimes typhoid or septic in nature, that give rise to the so-called "gastric fever." The term "*crapulous gastritis*" has been applied to those cases due to gluttonous meals. (2) Excessive indulgence in spirituous liquors is a common cause of acute catarrh of the stomach. (3) Acute infectious fevers, as measles, typhus fever, and scarlatina, provoke the disorder ("*erythematous gastritis*"), as do also remittent and intermittent fevers, especially when of the pernicious variety. (4) Certain drugs, as the salicylates and iodids, sometimes act as causative agents. (5) The influence of cold as an exciting factor of this disease has very probably been overestimated. (6) Since the publication of the observations by Klebs, Fränkel, and others the mycotic origin of the condition cannot any longer be doubted. It has long been known that the larvæ of certain insects may produce gastritis.

**Clinical History.**—The symptoms of the ordinary or milder variety of acute gastric catarrh are embraced in the description of the "sub-acute gastritis" or "acute dyspepsia" of some writers. Soon after eating there are uneasiness, fulness, pressure, distress, and, perhaps, a dull pain referred to the epigastrium. Thirst is common, also nausea, eructations of gas or liquid, and, less often, vomiting. The *vomit* consists of undigested food, considerable mucus, and fluid constituents that are sometimes bile-stained. The *tongue* is coated. The general condition of the patient remains unimpaired, and the average duration is less than twenty-four hours. In *severer cases* the symptoms before stated are intensified, and particularly the nausea and vomiting. Physical exploration discloses slight prominence of the epigastric area, with more or less tenderness on palpation. The *tongue* is dry and heavily coated, the breath unpleasant as a rule, the patient complaining of a flat or bitter taste in the mouth. *Constitutional symptoms* appear early, and the onset is often marked by rigor and a febrile reaction, the temperature rising to 102° F. or even 103° F. (38.8°–39.4° C.). *Herpes* may appear on the lips and skin—a fact that points to the infectious nature of this complaint. The *pulse* is usually accelerated, and there are indisposition to exertion, headache, dulness, and other nervous symptoms. An erythematous cutaneous eruption is often present, and particularly in febrile cases in children. That the marked disturbances of the general health are due to the toxic effects of the products of fermentation and decomposition is quite probable.

**Complications.**—Constipation is a comparatively frequent compli-



cation, and diarrhea a comparatively infrequent one. Either coincidentally or by direct extension the duodenum is similarly affected, and in some instances jaundice becomes an accompanying feature. The duration of this variety of the disease rarely exceeds four or five days.

**Diagnosis.**—The diagnosis of the lighter, afebrile forms of the disorder is not attended with the slightest difficulty. On the other hand, the diagnosis in cases in which well-marked local and general symptoms appear is not easy. The definite etiology, the vomiting, the pain or tenderness, the sudden rise of temperature, and the equally sudden fall at the end of a few days, are almost unequivocal.

**Differential Diagnosis.**—The absence of prodromata, of rose spots, of the peculiar temperature-range, and of enlargement of the spleen serve to distinguish this complaint from *typhoid fever*. The instances of indeterminate etiology may present a clinical picture not to be differentiated from certain infectious diseases. Here a careful analysis of the local symptoms and signs will usually lead to a correct conclusion, despite the apparently complete identity of the general disturbances. Close observation of the behavior of any given obscure case for a couple of days will usually enable the physician to arrive at a correct diagnosis. In children headache and vomiting are symptoms often so well marked as to create a striking resemblance to *tubercular meningitis*, but the latter can be discriminated by the history and longer duration. In children acute gastritis with an erythematous rash is often mistaken for *scarlet fever*. The final elimination of the latter disease is usually easy, however, in consequence of the absence of angina, of the typical tongue, the hard and very rapid pulse, and the peculiar desquamation affecting the hair and the nails.

**Prognosis.**—Quite generally the prognosis is good. When, as sometimes happens, however, the disease is purely secondary, the prognosis must depend largely upon the primary affection. I have found that many persons suffer from repeated attacks of gastric catarrh, each increasing the liability to subsequent attacks.

**Treatment.**—Our chief aim should be to remove the cause and then to give the stomach complete rest. Hence, whenever the disease is distinctly traceable to errors of diet, emetics of the blandest sort should be employed; large draughts of warm water usually suffice, but lavage is to be preferred in some cases. This should be followed by a purge made up as follows:

Ry. Hydrarg. chlorid. mit.,	gr. j	(0.0648);
Sodii bicarb.,	gr. xvij	(1.16);
Sacchari lactis,	gr. xij	(0.777).
M. et ft. chart. No. vj.		

Sig. One, dry on the tongue, every hour; the last to be followed in two hours by a wineglassful of Hunyadi Janos or other saline laxative.

The stomach must now have absolute rest for about twenty-four hours, when pancreatized milk or milk boiled with lime-water may be given at stated intervals. If nausea and continued vomiting prohibit the use of milk by the mouth, I resort to rectal alimentation early, and particularly in children. Certain symptoms, as *nausea*, *pain*, and *rest-*

*lessness*, demand as early relief as possible, and can be most successfully met by the use of morphin in small doses hypodermically at intervals of twelve hours. When constant nausea is the symptom chiefly complained of, I have found creasote combined with bismuth or cocain in small doses to be highly serviceable. Convalescence is usually uninterrupted, and is soon complete. When protracted it is often on account of the too early return to solid articles of diet or the too early use of bitter tonics. The mineral acids should first be administered, well diluted, after the local symptoms have in a great measure subsided, and to these the bitter vegetable tonics are later to be gradually added. Locally, I employ sinapisms at the beginning of severe types of the affection, and follow these with warm linseed poultices lightly applied to the entire epigastric and hypochondriac regions.

#### TOXIC GASTRITIS.

**Pathology and Etiology.**—This is an intense form of acute gastritis, produced by the ingestion of irritant and corrosive poisons, among the former being such agents as phosphorus, antimony, and arsenic, and among the latter concentrated mineral acids and strong alkalies. When caused by the non-corrosive poisons intense hyperemia and tumefaction, leading to desquamative changes in the glandular structure, ensue. When excited by corrosive substances necrosis of the mucous membrane may occur, leading even to an involvement of all the coats of the stomach-walls, and terminating in perforative peritonitis. The lesions may be of various grades of severity, and are either strictly localized or more or less general.

**Symptoms.**—The symptoms vary somewhat with the nature of the special poison, though they are usually quite violent. *Incessant vomiting*, great pain in the epigastric region, and, later, *diarrhea*, and excessive thirst, together with such symptoms as intense *burning pains* in the mouth and throat and dysphagia, are the most characteristic signs. The *vomit* contains mucus, sometimes blood, and rarely shreds of mucous membrane. The *physical examination* reveals a marked distention of the abdomen, which is also, as a rule, very painful on pressure over the epigastric region. The *general condition* of the patient soon becomes one of profound prostration; the skin-surface is cold and clammy, and the pulse and respiration are greatly hurried, terminating at times in fatal collapse within a few hours. Sometimes a marked febrile movement precedes the development of the symptoms of collapse. The temperature may reach 104° F. (40° C.); the pulse ranges from 100 to 130; and the urine may be scanty, containing a slight amount of albumin or red blood-corpuscles. As a sequel we may have symptoms of gastric ulcer or of esophageal stricture.

**Diagnosis.**—The diagnosis rests upon the history of the ingestion of some toxic material, upon the character of the symptoms (referable not only to the stomach, but also to the mouth and to the pharynx), and upon the results of the inspection of the mouth, pharynx, and the matters vomited. A chemical examination of the latter may be necessary.

**Prognosis.**—This depends upon the nature of the poison and its dose. When free emesis occurs early the prognosis is thereby rendered

more favorable, since under these circumstances both the local and constitutional effects are mitigated. Among unfavorable symptoms may be mentioned the development of signs of collapse or of peritonitis.

**Treatment.**—To ascertain, in the first place, the special cause of the gastritis, and when this is found to administer the proper antidote to that poison, are measures of prime importance. The stomach should be washed out with warm water containing some demulcent substance and a small proportion of the appropriate antidote. While lavage may be resorted to, it must be cautiously undertaken. Subsequently measures should be employed to combat the active local inflammation. Externally, leeches, followed by the ice-bag, have proved to be the best agents in my own hands; internally, opium, bismuth, and demulcents, with bits of ice, are most useful. Rectal alimentation should form the sole method of feeding so long as the signs of severe inflammation along the upper alimentary tract are present. The indications presented by the general conditions will vary with the general effects of the peculiar poison in each case.

#### DIPHTHERITIC GASTRITIS.

This form of gastritis is always a secondary condition, though it is not, as has often been stated by others, always caused by a direct extension of the diphtheritic membrane from the pharynx down through the esophagus to the stomach. It arises more frequently in the course of some other acute infectious malady, as pneumonia, scarlet fever, or small-pox. Though it is regarded as a rare disease, the fact that it is unrecognizable during life renders it certain that the affection is sometimes overlooked. I have seen two instances associated with croupous inflammation of the intestines, both occurring in greatly debilitated children. Osler saw a case which occurred as a secondary process in pneumonia.

#### ACUTE SUPPURATIVE GASTRITIS.

(*Phlegmonous Gastritis.*)

**Definition.**—An acute suppurative inflammation of the submucosa.

**Pathology and Etiology.**—Phlegmonous gastritis is confessedly a rare, and almost invariably a secondary, disease. I have observed pathologic evidences of its presence, however, in two cases that came to autopsy, both patients having died of sepsis. In general, the etiology is obscure. It may very rarely originate spontaneously; it may also follow an injury, though more commonly it is merely a symptom of a general septic process or a complicating condition of an acute infectious malady. Two forms are described—namely, a *diffuse purulent infiltration* and a *circumscribed form*. The morbid process begins in the submucous layer, and then spreads in various directions, involving soon the muscular and serous coats on the exterior and the mucous coat on the interior. The limited variety results in the formation of true abscesses, that may attain considerable size and rupture either into the peritoneal cavity or into the stomach.

**Symptoms.**—There may or may not be an initial rigor. Whether the case is ushered in by a chill or not, however, the *temperature* soon



risers to 103° or 104° F. (40° C.), and subsequently pursues an irregular course. The symptoms of the typhoid state supervene, and are usually associated with the symptoms of the primary affection. Hence the clinical picture is greatly diversified. For a variable period prior to the fatal issue the patient passes into coma. The local symptoms and physical signs are rarely diagnostic. There is a constantly increasing epigastric pain; emesis also appears, the vomita often containing a notable quantity of pus-cells.

The **physical signs** reveal but little in most instances, and vary with the form of the complaint. Inspection shows in the *diffuse* form a considerably distended abdomen. On pressure the stomach is found to be quite tender. In the *limited variety* the gastric abscess sometimes gives rise to the physical signs of a tumor, and a localized prominence may be seen over the seat of the abscess; the tenderness to the pressing finger may be confined to the same area. Palpation has served to elicit fluctuation and to define the limits of the tumor, the latter sometimes attaining the size of a cocoanut; on percussion either dulness or a muffled tympanitic resonance is elicited, according to the size of the tumor.

**Diagnosis.**—The diffuse variety cannot, as a rule, be positively distinguished from certain other gastric affections. The detection of pus-cells is, however, of the utmost diagnostic value. Gastric abscess, on the other hand, is often recognizable, since the physician has not only the history to aid him, but also the physical signs, which may demonstrate the presence of a fluctuating tumor.

**Course and Prognosis.**—The majority of cases reach a fatal termination within one week, and those that do not terminate in death thus early merge into a subacute or even chronic course. They present such symptoms as local pain, chills, and fever, and death results, sooner or later, either from exhaustion or such complications as peritonitis and metastatic abscess with jaundice.

**Treatment.**—The treatment in the diffuse form is, at best, only palliative. In the circumscribed variety the aid of the surgeon should be invoked as soon as a probable diagnosis has been made.

#### CHRONIC CATARRHAL GASTRITIS.

(*Chronic Catarrh of the Stomach; Chronic Catarrhal Dyspepsia.*)

**Definition.**—A chronic catarrhal inflammation of the gastric mucous membrane, presenting various degrees of intensity and embracing the symptoms that are more or less characteristic of widely different clinical forms of gastric derangement.

**Pathology.**—The anatomic changes are most marked near the pylorus, where the mucous membrane often presents a distinctly wrinkled, mammillated appearance. The mucous membrane looks either red or gray (the latter hue being due to pigmentation), and is pretty generally covered by tenacious mucus, mingled with detached epithelium. Ewald describes the histologic changes thus: "The minute anatomy shows the picture of a parenchymatous and an interstitial inflammation. The gland-cells are in part eroded or show cloudy, granular swelling or atrophy. The distinction between the 'haupt' and 'beleg' cells cannot be recognized, and in many places, particularly in the pyloric region, the

tubes have lost their regular form and show in many places an atypical branching like the fingers of a glove. Individual glands are cut off toward the fundus, but appear at the border of the submucosa as cysts, partly empty, with a smooth membrane, partly filled with remnants of hyaline and refractile epithelium. An abundant small-celled infiltration presses apart the tubules, and is particularly marked toward the surface of the mucosa, and from the submucosa extensions of the connective tissue may be seen passing between the glands. The mucoid transformation of the cells of the tubules is a striking feature in the process and may extend to the very fundus of the glands." Hemorrhagic abrasions may be found in cases due to cardiac disease or to portal engorgement. If the catarrh is of long duration, further anatomic changes occur. Superficial ulcers may form, usually in the pyloric region or along the lesser curvature, varying in size from a few lines to an inch or more in diameter, and nearly circular in shape. Long-standing cases also present sclerotic changes of the mucous membrane. Of these, two forms are distinguished. In the one variety the mucous membrane is perfectly smooth and atrophied; the glands are displaced, narrowed, and shortened, while the gap thus formed is more or less filled with connective tissue. There is a thinning of the stomach-wall, with enlargement of its cavity. The other form presents a hyperplasia of the mucous membrane, the glandular structure, and the submucous layer, sometimes resulting in enormous thickening of the stomach-walls, with great diminution in the size of its cavity. I have seen one instance in which the stomach held less than a half pint at the autopsy.

**Etiology.**—It is evident that the factors which produce acute gastric catarrh will, if long continued, produce a chronic condition. The latter has many causes, all of which act either as mechanical, chemical, or biologic irritants, and fall naturally into the following classes: (*a*) Errors of diet (referring more particularly to improper articles of food), its variety, and preparation; excessive alimentation; the habit of eating at irregular intervals or with undue haste, and thus not allowing time for perfect mastication of the food. (*b*) The immoderate use of alcohol doubtless stands second in order of importance. Those persons who habitually indulge in alcoholic beverages to excess are prone to an irregular mode of life, and this of itself tends to produce debilitated states of the system that lead to digestive disturbances and reinforce the baneful effects of the excessive use of alcohol. Such patients are apt to suffer from the more active forms of the complaint, and, at shorter or longer intervals of time, from genuine acute gastritis (*vide* Etiology of Acute Gastric Catarrh). In the same category should be mentioned other gastric irritants, as the excessive use of tobacco and the prolonged use of tonics and purgatives. (*c*) Functional derangements of the stomach sometimes merge into the disease under consideration. This is especially true of that form in which there is a deficiency in the gastric juice. Under these circumstances fermentative and putrefactive changes set in, and the products thus generated become sources of chronic irritation. (*d*) Local mechanical influences may offer resistance or obstruction to the outflow of venous blood from the stomach to the right heart. These consist in portal congestions, either passive or more or less active according to their special causation. In this class of cases chronic gastric

catarrh is a secondary process in chronic affections of the liver, heart, and lungs. (c) Such constitutional conditions as gout, chronic rheumatism, chronic tuberculosis, Bright's disease, diabetes, anemia, chlorosis, chronic malaria, syphilis, and chronic forms of skin-disease. The explanation of the peculiar liability of these conditions to catarrh of the stomach lies in the obstruction offered to the passage of blood through the hepatic and cardio-pulmonary circulation. This is true in an especial degree in chlorosis, anemia, chronic tuberculosis, and malaria; in gout, chronic Bright's disease, and syphilis it is probably due largely to the action of chemico-vital irritants in the circulating medium.

**Clinical History.**—The *local symptoms* bear a striking resemblance to those of other forms of gastric disturbance. They vary greatly in severity, though never entirely absent, as in the case of purely functional disorders. Deficient secretion of the gastric juice, due to the anatomic changes in the gastric tubules, is a potent factor in the production of the symptoms directly referable to the stomach. It is the function of hydrochloric acid, normally present in the gastric secretions, to destroy the ferment-producing spores; hence when, owing to lack of free HCl, the latter are not destroyed, deleterious products of fermentation are the result, these in turn aggravating and prolonging the course of the affection. Recent investigations go to show that deficient motor power is more important than a deficiency in the secretions in bringing about the clinical phenomena of the disease. The presence of an inordinate amount of mucus which is alkaline in reaction neutralizes in part the HCl; it may also more or less completely cover the ingesta, thus preventing the gastric secretions from reaching them, and lengthening, at the same time, the period of digestion.

Among the *earlier symptoms* directly attributable to the gastric lesions are anorexia (though at times the appetite may be moderately good or even keen); fulness and distress; burning sensations and dull pain in the epigastric region; eructations of gas, which may be either offensive or odorless, during and immediately after meals; regurgitation of fluid, either acid (heartburn), due to the presence of organic or hydrochloric acid, or a bitter form of peptones. These symptoms are usually increased in intensity after meals. The *tongue* frequently appears broad and flabby, and almost constantly the edges and tip are somewhat reddened, whilst the papillæ are enlarged. Occasionally it is small, with enlarged and red papillæ, or it may look healthy. A bad or, at times, a persistently bitter, taste in the mouth and great thirst may be complained of. There is often a profuse secretion of saliva, but the mouth may be dry. *Nausea* is common, and is most marked in the morning hours; it is frequent before or after meals, and often *vomiting* occurs either immediately after meals or a couple of hours later. The *vomit* will vary somewhat with the time of the occurrence of emesis. Usually it consists of food in the first stages of digestion, mixed with large quantities of mucus. In alcoholic catarrh morning vomiting occurs quite commonly, and consists of a watery fluid composed of saliva and mucus. This class of sufferers from chronic gastritis not infrequently exhibits well-marked evidences of salivation. I have repeatedly found the material vomited in chronic gastric catarrh to be acid in reaction, unless, as occasionally happens, the vomiting takes place several hours



after eating, when it is sometimes faintly alkaline or neutral. The acidity of the vomitus is not due solely to the presence of HCl, but partly and sometimes largely to acid salts or the weaker acids (lactic, butyric) resulting from the abnormal processes of fermentation previously mentioned.

*Microscopic examination* sometimes reveals the presence of sarcinæ ventriculi, yeast fungi, and numerous bacterial organisms. The relations of these low forms of life to the pathologic processes going on in the stomach are not well understood, except in the case of the yeast fungus, which is concerned with the process of fermentation. The sarcinæ ventriculi may, however, exercise a causative influence, since certain cases yield readily to the antiseptic method of treatment.

A *chemical examination* of the contents of the stomach for purposes of diagnosis according to the methods laid down in the preliminary section (*vide* p. 735) should not be neglected. In simple chronic gastric catarrh the hydrochloric acid is found to be diminished, and sometimes, though not as a rule, lactic, butyric, and acetic acids are present. In aggravated forms this result may be modified by the presence of the organic acids and by a great relative diminution in HCl. In many cases of chronic catarrhal gastritis there is an abundance of mucus, and an excess of hydrochloric acid may be met with in proliferative gastritis. On the other hand, in atrophic gastritis there is little or no mucus in the gastric contents, while there is in established cases an absence of HCl and of the digestive ferment. Ewald has subdivided all cases into three varieties: (*a*) *Simple gastritis*, in which the fasting stomach contains only a small quantity of slimy fluid, while after the test-breakfast the HCl is diminished in quantity, and lactic acids and the fatty acids are usually present. (*b*) *Mucous gastritis*, in which class the acidity is always slight and the condition is distinguished from simple gastritis by the large amount of mucus present. (*c*) *Atrophy*. Here the fasting stomach is always empty, while after the test-breakfast HCl, pepsin, and the curdling ferments are wholly wanting.

The *absorbent* and *motile* powers of the stomach are both more or less diminished, and are determined by the potassium iodid and salol tests respectively.

**Physical Signs.**—Sometimes there may be observed an undue distention of the stomach, the prominence being more marked toward the left. On making *firm pressure* over the epigastric region tenderness is often elicited. This is not present in the early stages, nor constantly later in well-marked cases, since the degree of inflammatory action is subject to great oscillation. When tenderness is found over a considerable portion of the epigastrium in the absence of a new growth, it is of great diagnostic value. It is to be recollected, however, that resistance may be felt when the stomach is thickened in chronic interstitial gastritis. Dilatation of the organ may be indicated by splashing sounds, and the latter may be elicited in the absence of gastrectasis at a time when the stomach should contain no food.

On *percussion* there are discoverable alterations in the size of the organ.

Among the general or indirect symptoms manifested the *nervous phenomena* are of first importance. So prominent are they in the clinical

picture oftentimes as to lead the incautious physician to the conclusion that his patient is suffering from some primary disease of the brain or nerves. The nervous derangements have been attributed solely to morbid sympathetic disturbances; it is quite probable, however, that we should ascribe a share of the morbid influence to the absorption of toxic materials from the stomach and intestines. Headache is frequently complained of; it is generally frontal, though also occipital, and tends to appear before meals. The so-called sick headache more rarely occurs. Indisposition to mental or physical exertion, vertigo, depression of spirits, and well-marked hypochondriasis are common concomitants. Patients complain of wakefulness and disturbed dreams, though drowsy after meal-time. There is a sympathetic disturbance of the cardiac rhythm, and sometimes dyspnea, owing to the same cause. The urine is often highly colored, scanty, and deposits an abundant uratic sediment; occasionally, however, it is of low specific gravity, rather copious in amount and pale in color, owing to the influence of phosphates. This condition is found in cases in which the nervous element is notably prominent.

**Complications.**—The intestines often become involved, and usually by direct extension. Implication of the duodenum may lead to jaundice and to obstinate constipation, though only moderate constipation is the rule in catarrh of the stomach. When the process extends to the large intestines diarrhea develops. Alternating constipation and diarrhea are often observed. The nutritive system is, in confirmed cases, seriously implicated, as shown by the anemia (of the pernicious type in some cases), emaciation, and general debility present. It is particularly in examples of combined intestinal and gastric catarrh that we observe the most notable impairment of the general health, and the reason of this will be clear when it is recollected that under these circumstances all the digestive fluids are lessened in amount. The gases generated in the stomach often find their way into the intestinal canal, giving rise to tympanitic distention, and sometimes to colicky pain. In not a few instances the gastric catarrh extends upward to the oral cavity. Under such circumstances the tongue is large and heavily coated, with impressions of the teeth upon its edges. Since the mucous membranes are unhealthy, there is produced an abnormal condition of the secretions that renders the breath foul and causes thirst. Certain skin-eruptions, as eczema, lichen, and urticaria, are common concomitants of this disease. By some authors these disorders of the skin are supposed to be caused by the catarrh of the stomach. I have frequently observed, however, that when present their improvement has been followed by an aggravation of the gastric symptoms, and *vice versâ*. A sequel of the disease is dilatation of the stomach, but I believe this to be less frequent than was formerly supposed. The course of chronic gastric catarrh is long, the average duration being considerably more than one year. Its duration may be much abridged by early recognition and proper treatment of the condition. The symptoms at first intermit and are mild, but later are persistent.

**Diagnosis.**—A positive diagnosis may be based on a clear etiology, the presence of persistent symptoms and signs of chronic disturbance of digestion, upon defective motor power, a diminished amount of HCl, an abundance of mucus in the gastric contents, and deficient absorptive power. The points of difference between the more serious affections of

the stomach (carcinoma, ulcer, and dilatation) and chronic gastric catarrh will be detailed when the former diseases are considered. As I have said, Ewald makes three leading forms of the complaint, based on the results obtained from an analysis of the contents of the stomach, but I have found his classification not satisfactory in practice.

**Prognosis.**—Chronic catarrh of the stomach may be said not to manifest an innate lethal tendency. It, however, predisposes to, as well as aggravates, the symptoms of existing forms of acute and serious forms of chronic diseases, especially organic affections of the stomach. The prognosis depends considerably upon the stage in which the disease is when first met with, since the condition is amenable to treatment only when not too far advanced. The prognosis is rendered somewhat more grave by the presence of certain complications previously mentioned. I have seen one case that proved fatal in consequence of stricture of the pylorus.

**Treatment.**—It must never be forgotten as far as possible to search for and remove the causal affections in every case. When associated with grave forms of cardiac, hepatic, or renal disease these must receive careful attention primarily.

The masticating apparatus must be looked after by the physician, who must also instruct his patient in the art of eating slowly, so that insalivation of the food is thoroughly effected. Too often the quantity of aliments consumed is beyond the needs of the bodily functions, and the method of preparing the same faulty—defects to be early corrected. Such patients should eat oftener than in health, taking four or five meals in the twenty-four hours. The physician must with untiring diligence attend to every dietetic, sanitary, and therapeutic detail. The major portion of the treatment has relation to—

(1) *The Diet.*—In the matter of arranging the dietary in separate cases the general condition and peculiarities of the individual must be taken into account. The wise physician will be guided to some extent by the dictates of his patient's experience, and will not fail to avail himself of any information obtainable upon this head. In *severe cases* an exclusive milk diet for a period of two to four weeks often gives the best results. The daily amount requisite to meet the demands of the vital functions is 4 to 8 pints. Of this, 5 to 8 ounces are to be taken *slowly* every two hours during the day. The beginning amount, however, must occasionally be smaller—2 to 3 ounces—to be gradually increased. A pinch of salt or from  $\frac{1}{2}$  to 1 ounce of lime-water may be added to each feeding, or the milk may be diluted with Viehy. The milk should not be taken iced, but warmed or at the temperature of the room. Boiled milk is objectionable. The stools are to be watched for curds, and when the digestive capacity is exceeded the amount of the nutrient should be lessened and other articles cautiously added.

When *whole* milk cannot be digested on account of an actual loathing for it, skimmed or partly skimmed milk or buttermilk should be substituted. If the latter cannot be utilized in proper amount, animal broths, together with some of the artificial foods (panopeptone, liquid peptonoids), may be added. As tolerance for a liberal amount of milk becomes established the appetite is no longer satisfied, and then I begin to add the light solids in a gradual manner; for example, white meat of chicken or



game (except tame ducks and turkey), stale or twice-baked bread, milk or dry-toast or zweibaek, soft-boiled eggs, fish, and, later, Hamburg steaks, stewed sweetbread, and the like. For dessert, junket or custards, sweetened with saccharin, are well borne as a rule. Subsequently, farinaceous articles, if thoroughly cooked (except oatmeal), and certain plain vegetables, may be allowed, but their effects must be minutely observed. The former are to be eschewed in cases in which acid-fermentation or flatulency is a prominent feature. Among the latter, rice, spinach, lettuce, and macaroni (stewed in milk) are to be selected. Peas and beans, if green and succulent, may be tried, but if ripe are to be discarded. The only form of fat permissible is good butter. Stewed fruits are often well borne and tend to overcome constipation. In cases in which the latter symptom obtains Graham bread and green soft vegetables are also indicated and often are readily digested.

In light cases and in those of moderate severity, particularly if the cause of the complaint is removable, the dietary need not be rigid at the start. Indeed, to minimize the saccharine articles and starches and to avoid the coarser vegetables, hot bread, pastries, and the like, is all that is required. In the case of confirmed dyspeptics the following articles are to be scrupulously avoided: very fat meats, fat fish-foods, condiments, certain fruits (strawberries, bananas), hot bread, saccharine articles of diet and farinae, potatoes, particularly sweet potatoes, and other coarser vegetables.

Of *drinks*, the best during meal-time is simple hot water, to which a little milk may be added, or a single coffee-cup of weak tea. Occasionally cocoa is allowable, but ordinary chocolate, coffee, and strong tea are harmful. Too much liquid should not be taken during a meal, since it dilutes the gastric secretion to a deleterious extent, and cold drinks are to be interdicted during the same period. I have never observed any unfavorable effects from the moderate use of ice-water between meals. Alcohol, and particularly concentrated spirituous liquors, exert an irritating effect, and hence in cases of chronic catarrhal gastritis should be absolutely forbidden.

(2) *Hygienic measures* are of signal value in this disease. Of these the most important are forms of fresh-air exercise, as bicycling, walking, boating, and horseback-riding. Suitable indoor apparatus for physical exercise is now easily obtainable at little cost, and therefore open-air exercise may be supplemented by the latter. Physical exercise must be carefully supervised, so as to avoid the deleterious effects of over-exertion. I am convinced of the superior advantage of travel, including a sea-voyage, and an appropriate change of air—for example, to the seaside or mountains—particularly for the large class of self-centered and low-spirited dyspeptic patients. A cold sponge-bath, followed by brisk friction of the skin, is to be advised. An abdominal bandage, made of woolen or silk material and constantly worn, tends to increase the patient's comfort.

(3) *Medicinal Treatment*.—Saline laxatives, as sodium phosphate, Rochelle salts or Carlsbad salts, taken fasting in hot water, are advantageous, since they serve to regulate the bowels, to deplete the engorged gastro-intestinal vessels, as well as to rinse the stomach. Hunyadi Janos or Carlsbad mineral waters may be substituted. Their efficacy is much

enhanced in cases in which gastro-duodenal catarrh is associated with portal congestion, when the alkaline carbonates are administered simultaneously. Further than this, little is needed in the majority of instances. The use internally of antiseptics, combined with alteratives and mild astringents, is often beneficial. I can speak most positively in favor of the following pill:

R $\bar{y}$ . Argenti nitratis, gr. iv (0.259);  
 Ext. hyoscyami, gr. viij (0.518).  
 M. et ft. pil. No. xvj.

Sig. One about one hour before each meal, the stomach being first prepared by rinsing with a 2 per cent. solution of borax in water.

The silver nitrate is to be continued for a period of five to eight weeks, with interruptions of several days at the end of three weeks.

In the *fermentative* form of chronic gastric catarrh the hyperacidity is, in reality, often dependent upon the lack of free HCl; hence this agent should be supplied. It is best administered immediately after meals, the dose being not less than 10 minims (0.666), well diluted, and this may be repeated in the course of ten or fifteen minutes in obstinate cases; it may be combined advantageously with pepsin (gr. v-x—0.324–0.648) or pancreatin (gr. x—0.648). Pancreatin is better associated with sodium bicarbonate in the form of a tablet containing each gr. ij (0.129). Of these two or three may be administered fifteen to thirty minutes after meal-time. Care is to be taken to use only the best articles of pepsin and pancreatin. When hyperacidity exists, diastase and ptyalin may be exhibited, but I have failed to obtain encouraging results from their employ. This class of cases represents an aggravated or advanced form of the disease (atrophic stage), and demands prolonged and varied treatment. At the end of the digestive process it is well to thoroughly irrigate the stomach (lavage), and more particularly if evidences of dilatation be present. The stomach may also be cleansed and prepared for the reception of the next meal in a very agreeable manner by having the patient sip a 2 per cent. solution of borax in warm water or a 2 per cent. solution of sodium chlorid half an hour before meals; indeed, the continued use of simple hot water for the same purpose has, in my hands, often given excellent results. With it must, of course, be combined the saline laxatives and the restricted diet. Not less than 1 pint of water, hot as it can be taken by the patient, should be sipped at each sitting.

To assist the appetites of these patients and to stimulate the secretory function a few drops (not more than 5) of the tincture of nux vomica may be given fifteen minutes before meals, with gr. ij–iij (0.129–0.194) of sodium bicarbonate. These indications are also fulfilled by lavage once daily or bi-daily (if the patient be feeble). If hyperacidity, due to the organic acids, persists despite the measures already recommended, we may combine bismuth subnitrate with magnesia and a few grains of charcoal, this being administered when the stomach is empty. We may also check fermentation by the exhibition of salicylic acid (gr. v—0.324) thrice daily or creasote (gr.  $\frac{1}{2}$ —0.0324) thrice daily. Germain Sée has recently found strontium bromid (5ss to 3j; 2.0–4.0) to be of great value in cases in which gaseous fermentation with hyperacidity is combined with permanent ten-

derness. Happy results often follow a course at some spa if the patient be under the charge of a competent physician during his sojourn. The robust or plethoric should go to Carlsbad, Ems, and Kissingen abroad, and to Saratoga in this country, using more especially the Hawthorne water. The anemic should go to Franzenbad or the spa near Brussels, and in this country to the iron springs at Bedford, Pennsylvania. A course of the alkaline mineral waters may be successfully taken at home in many instances, though patients are much more apt to obey the physician's injunctions as to diet, exercise, and the like when at a spa than when at home. These waters do not simply act as purgatives, but also as antacids. It has been experimentally shown that sodium chlorid, sodium carbonate, as well as carbon dioxid, promote the secretion of the gastric juice. In the more chronic cases belonging to this class or those that have resisted other forms of treatment intestinal complications are usually found. Here the alkaline waters are to be alternated with calomel in small doses, prescribed thus :

R̄. Hydrarg. chloridi mitis,	gr. ij (0.129);
Sodii bicarb.,	ʒj (4.0);
Sacchari lactis,	ʒss (2.0).

M. et ft. chart. No. xij.

Sig. One, dry on the tongue, four times daily.

I have been in the habit of continuing the use of these powders for several days to one week, then returning to the alkaline waters for two weeks, and so on.

In the *mucous* variety of gastric catarrh additional indications for treatment are presented. The chief aim should be to limit, as far as possible, the production of mucus and to cleanse thoroughly the stomach prior to each meal, thus preparing the organ for the reception and better digestion of food. Here, again, at least one pint of hot water, containing the substances before mentioned, should be sipped half an hour before each meal. This mode of cleansing the stomach is usually successful; if unsuccessful, however, it should be supplemented by lavage once daily, using the same solutions as above indicated, though in larger quantities. The siphon is also highly useful in cases of this sort in which stricture of the pylorus is suspected and when the food is retained in the stomach much longer than the normal period of digestion. This frequently happens, for the reason that the mucous covering which the food receives not only prevents it from being acted upon by the gastric juice, but also renders absorption tardy. The therapy of this form of chronic gastritis requires, in addition to what has before been given, the more potent astringents for the purpose of arresting hypersecretion of mucus. The best way to use these agents is topically. The stomach may be washed out (at bed-time or early in the morning) with a 2 per cent. solution of alum or a 1 per cent. solution of tannic acid; antiseptic solutions are employed in like manner, a 2 per cent. solution of salicylic acid being especially efficacious. If lavage cannot be practised, such astringents as catechu, cerium oxalate, and silver nitrate, with small doses of opium (*vide supra*), should be tried. For use internally one of the very best remedies is atropin sulphate.

*Certain symptoms* belonging to all varieties of the affection may demand



relief. These must be met in accordance with general principles. Vomiting, which is at times a distressing symptom, is best allayed by small doses of resorcin or creasote in combination with cerium oxalate.

As soon as the morbid irritability of the stomach has been reduced mild forms of bitter tonics, with a view to imparting vigor to the digestive organs, may be cautiously employed. Their too early use is very apt to aggravate existing symptoms, or even to reproduce such as have already disappeared. Iron is often indicated during convalescence.

## GASTRIC ULCER.

(*Simple or Round Ulcer of the Stomach.*)

**Definition.**—An ulcer presenting sharp borders, with a tendency to extend in depth, generally without collateral inflammation, giving rise, usually, to one or more characteristic symptoms, as pain, vomiting, and hematemesis. Rarely it is entirely latent.

**Pathology.**—The gross anatomic characteristics and peculiarities may be briefly considered seriatim. (*a*) In *shape* it is usually round or oval. It rarely happens that there are several ulcers, and these may form larger ones having irregular borders. They are at first superficial, though their floor (when seen at autopsy) is below the mucous membrane, owing to a tendency to extend in depth. This characteristic has given rise to the term “perforating ulcer.” Thus, the ulcer has for its base, very frequently, the muscular or serous coats, but sometimes, and not rarely, the ulcerative process extends through the walls of the stomach, in which case adhesions form between the stomach and the adjacent viscera, one or other of the latter organs occupying the base of the ulcer. Almost always the walls slope inward, giving rise to the characteristic funnel-shape. The edges may, however, be sharp and abrupt. The floor of the ulcer is quite generally clean, and rarely may present a hemorrhagic aspect. A recent ulcer presents clean-cut edges, that are not the seat of collateral inflammatory edema, though an old ulcer often presents somewhat thickened margins. (*b*) In *size* it is quite variable. The majority of the ulcers are not larger than a dime; others may measure as much as 10 cm. (4 inches) in their greatest diameter. The edges are almost invariably formed from the coalescence of two or more smaller ones. (*c*) The *position* is most frequently near the pylorus on the posterior wall, and particularly in the vicinity of the lesser curvature.<sup>1</sup> Fortunately, they occupy the anterior surface but rarely, this being a dangerous situation, as will presently be explained.

The ulcer often heals by cicatrization. The resulting scar is pale and stellate, and there is puckering of the surrounding mucous membrane. If the ulcer has not extended deeper than the mucous membrane, granulation-tissue develops from the edges and base; this tissue slowly con-

<sup>1</sup>Of 793 cases collected by Welch from hospital statistics, 288 were on the lesser curvature, 235 on the posterior wall, 95 at the pylorus, 69 on the anterior wall, 50 at the cardia, 29 at the fundus, 27 on the greater curvature. The duodenal ulcer is usually situated just outside the ring in the first portion of the gut (Osler, page 369).

tracts, uniting the margins and leaving a comparatively smooth scar. On the other hand, if the ulcer be large and involve the muscular and serous coats, stricture of the pylorus, followed by dilatation, may result. The stomach may present an hour-glass shape, due to the contraction of a girdle ulcer in the central part of the organ. Nearly all gastric ulcers would perforate the coats were it not for the development of a local peritonitis which establishes adhesions between the corresponding portion of the stomach and adjacent structures. The ulcers being usually situated on the posterior wall, the surface of the pancreas forms the point of attachment most frequently, though the stomach may also become adherent to the left lobe of the liver, the spleen, omentum, diaphragm, or the transverse colon. The organs with which the stomach becomes agglutinated may be penetrated by the ulcerative process, resulting in suppurative inflammation; or fistulous connections of the stomach with the transverse colon, the pleura, the pericardium, lungs, gall-bladder, and the duodenum may thus be established. Of these, gastro-colic fistulæ are the most common. Osler states that there are two instances on record in which the ulcer perforated the left ventricle. Penetration of the ulcer through the posterior gastric wall opens the lesser peritoneal cavity, in which case the base remains limited, producing a condition known as subphrenic pyo-pneumothorax. When the anterior surface of the stomach, which has no anatomic relations with other organs favorable for the establishment of protective adhesions, is perforated, general infectious peritonitis rapidly supervenes if a fatal end be not reached immediately. Intense hyperemia or the erosion of small vessels gives rise to small or moderate hemorrhages. If the ulcer penetrate one of the larger vessels, as happens not rarely, then profuse and even fatal hematemesis is the result. This accident is doubtless frequently prevented by the development of a "protective thrombosis." In several instances small aneurysms have been found at the bases of the ulcers (Douglas, Powell, Welch).

**Etiology.**—Since gastric ulcer was first accurately described by Cruveilhier many and widely various theories as to its mode of origin have been promulgated. Whilst there is to-day no universally accepted view of its pathogenesis, yet there are two points that may be regarded as definitely settled: (*a*) that the ulcer is due to a self-digestion of a circumscribed portion of the stomach; (*b*) that the alkalinity of the part digested has been previously reduced. Among the conditions lessening the supply of alkaline arterial blood, which, as is well known, prevents the stomach from being digested in health, the chief are embolism and thrombosis of the nutrient artery of the part, the infarct thus produced being annihilated by the gastric secretions (Virchow). This view receives confirmation from the fact that many instances of gastric ulcer have been observed in connection with disease of the cardio-vascular system. Opposed to this view are the experiments by Panum and Cohnheim, which show that ulcers produced artificially by occluding the arterioles with emboli tend to heal rapidly. Without stopping to detail all the other theories that have been propounded to explain their mode of development, I will mention only a few additional, and probably predisposing, causes. Most influential among them stands, doubtless, hyperacidity of the gastric juice—a condition almost universally present in this disease; although the

ulcers may not result primarily from the presence of an excess of acid, it is quite probable that further extension of the ulcerative process may be due to this factor. Peter assumes the cause of simple ulcer to be gastritis, and it cannot be gainsaid that the former is often met with in connection with the latter disease. On the other hand, Stockton holds the disease to be a neurosis. It is well known that the affection is often secondary in chlorosis, anemia, and oftener still in amenorrhea. The fact that in all the different forms of anemia there is a diminished alkalinity of the blood is of great interest in this connection. Obviously, then, ulcer of the stomach occurs more frequently in females than in males. It is most common between seventeen and thirty-five years; it is rare in young children, though Gorgart saw an instance in a child thirty hours after birth, and less rare in those past middle life. It is more frequent in the poor than in the rich: occupation has also a noticeable influence, and I have personally seen a number of instances in weavers. It is also prone to attack servants, cooks, and needlewomen among females, and shoemakers and tailors among males. Injuries of the immediate vicinity of the epigastrium have been often followed by gastric ulcer, but these have, as a rule, healed rapidly, and are not to be classed as "peptic" ulcers.

**Clinical History.**—In *typical cases* of gastric ulcer the clinical symptoms are almost positively diagnostic. The earliest symptoms point, very frequently, to chronic or subacute gastric catarrh, these being followed, soon or late, by those that are characteristic, as *pain, vomiting, and hematemesis*. Of these, pain is most constantly present, and presents certain peculiarities that demand rather elaborate mention. It is commonly dull, at times burning, and is associated usually with great oppression. These symptoms are doubtless often due to coexisting catarrhal gastritis. The character of pain that is most diagnostic is an *intense gnawing, burning or boring* in the epigastrium, more or less *periodic and strictly localized* in a circumscribed area. These paroxysms usually come on almost immediately after eating, occasionally one or two hours later, and disappear quite promptly when the stomach is emptied either by vomiting or by its contents passing into the duodenum. From the time of its occurrence, the quality, and strict localization of the pain, it may safely be assumed that it is due to direct irritation, set up by the food, of the sensory fibers occupying the base of the ulcer. In addition, there are paroxysms of diffuse pain (gastralgia) that are often strictly intermittent, though not necessarily excited by the partaking of food. This pain is due to a sympathetic nervous disturbance or reflected irritation. Finally, sharp, intense, lancinating pains, that are caused by local or general peritonitis, may appear suddenly, ceasing only with the death of the patient. We often meet with the four kinds of pain above described in a single case, though they vary in relative intensity in different cases. The pain in round gastric ulcer is greatly modified by numerous conditions, all of which are largely under human control. The *effect of taking food* has been already referred to, though it should be added that, obviously, undigestible, imperfectly masticated, highly-spiced food, sweet and hot substances, cause the paroxysms to be more intense than less irritating articles of diet. *Rest* diminishes the severity of the pain in that it prevents traction on the ulcer. *Certain postures* may aggravate it, and, though not a trustworthy guide, we may often de-



termine the situation of the ulcer by the effect of posture after taking solid food. The severity of the pain is often increased by bodily fatigue or even moderate exercise, and, to a greater degree, by special emotional influences. The situation of the pain when strictly localized is of the utmost importance in diagnosis. I have found it invariably from one to two inches below the ensiform cartilage, though it has been observed in the umbilical and hypochondriac regions. It is absent in one-half of all cases.

*Vomiting*, next to pain, is the most frequent symptom, but unless the vomitus contains blood, which is present in less than 50 per cent. of all the cases, it has little diagnostic value. Nausea and eructations of acid or food often precede or accompany the emesis. Vomiting usually occurs about two hours after eating, and is often coincident with the height of the paroxysm of pain, which the vomiting relieves as a rule. The *vomitus*, as first shown by Riegel, usually contains an increased proportion of HCl.

*Hematemesis* is a symptom of unequalled clinical significance. On it alone frequently rests a positive diagnosis. When the hemorrhage is considerable, pure blood, more or less clotted, may be ejected, this being highly characteristic of gastric ulcer. Frequently, however, the blood oozes gradually into the stomach and mingles with the gastric juice, and in consequence the oxyhemoglobin of the blood is converted into hematin, the vomitus presenting the appearance of coffee-grounds. On *microscopic examination* under these circumstances only larger or smaller pigment-masses can be seen, and no blood-corpuscles.<sup>1</sup> Vomiting of blood may recur at intervals of a few hours or on each successive day. The *amount* also varies within the widest limits according to the size of the vessel eroded. Some of the effused blood passes through the pylorus, escaping with the feces and giving to the latter a tarry, black appearance. A few cases have been reported in which all the blood was evacuated with the stools except that which was absorbed from the alimentary tract. Either as the result of a single copious hemorrhage or of repeated smaller hemorrhages a *pronounced anemia* is produced, the objective signs and the cerebral and cardiac manifestations of the latter disease at once becoming evident. As a rule, however, the evidences of anemia are only moderately well marked, and to assume that the anemia is due solely to the hemorrhages would probably be an error. A slight rise of temperature is often observed under these circumstances; this is to be regarded as the so-called *anemic fever*. Beneficial effects often follow hematemesis. The pain and the most unpleasant local symptoms have been frequently observed to disappear after its cessation—a circumstance that, as Strümpell observes, may be owing in part to the extreme caution of the patient thereafter. Not infrequently convalescence sets in almost immediately.

**Physical signs** are few and slight. On *palpation* tenderness is found, though not in all cases. The spot of localized agonizing pain before alluded to is often excessively tender on pressure—a valuable sign. The true gastralgic attacks, so common in gastric ulcer, are at times relieved by making firm pressure with the broad hand over the epigastrium. Near the pyloric end of the stomach palpable tumors may be felt, due to the

<sup>1</sup> The blood, however, can be identified by chemical tests and the spectroscopic appearance of the hematin.

thickened floor of the ulcer. When these indurated masses become adherent to adjacent organs—the pancreas, for example—epigastric tumors of considerable size may be felt, suggesting the presence of carcinoma. *General symptoms* often do not appear until late in the disease, the patient continuing to look as well as usual. Anemia is usually noted first, to be followed by debility and emaciation; the degree of the general disturbances is in direct proportion to the severity and duration of the causes producing them—namely, the coexisting catarrh, hemorrhages, pain, and vomiting. In some instances the cachexia is pronounced, and the face, on account of the prolonged suffering, assumes a drawn, haggard appearance.

**Other Clinical Forms.**—These have been subdivided into numerous types, some of which merge into one another and cannot be separated clinically. The following atypical forms should be distinguished: (a) Latent ulcers, whose existence is not suspected during life, but which are revealed, should they come to autopsy, as open ulcers or cicatrices. (b) An explosive form, in which the ulcer may or may not give rise to gastric disturbances prior to the occurrence of perforative peritonitis. (c) A recurrent form, described by Welch thus: “In this the symptoms of gastric ulcer disappear, and then follow intervals, often of considerable duration, in which there is apparent cure, but the symptoms return, especially after some indiscretion in the mode of living. This intermittent course may continue for many years. In these cases it is probable either that fresh ulcers form or that the cicatrix of an old ulcer becomes ulcerated.”

**Complications.**—Perforation of the ulcer leads to peritonitis, which almost always ends fatally. In exceptional instances, however, a localized peritonitis is the result, owing to rapidly forming adhesions or perforation into the lesser peritoneal cavity. The symptoms of this complication will be given in their proper place. Hemorrhage may prove a serious complicating accident, being in not rare instances an immediate cause of death. I recently saw a case of gastric ulcer in which hemorrhage was followed by rapid dissolution.

**General Course.**—This presents wide variations in different cases. It may be, though seldom, limited to a few hours, as in the explosive form. Innately, the disease is an exceedingly chronic one, often lasting several, and sometimes ten or fifteen, years. Its duration in curable cases may be lessened by proper treatment. Recovery may be incomplete, and the scar resulting from the healing process may give rise to true attacks of gastralgia. Again, if the cicatrices be situated at the pyloric orifice, dilatation will almost invariably develop.

**Diagnosis.**—The typical cases in which the characteristic symptoms above mentioned are conspicuous are easy of diagnosis. Hemorrhages occurring with gastralgic attacks are almost pathognomonic. A considerable proportion, however, offer great difficulties. Without the presence of hematemesis, for example, a positive diagnosis should not be made, and yet this symptom does not appear in 50 per cent. of all cases. In the absence of hemorrhage we may, however, infer the altogether probable existence of ulcer if there be a history of the more important etiologic factors; if there be gastralgia, hyperacidity, local pain, and tenderness; and, particularly, if the latter symptoms be excited or greatly aggravated by the taking of food. The long course and liability

to remission, to be followed by exacerbations of the symptoms, are strongly confirmatory.

**Differential Diagnosis.**—This disease may be mistaken for gastralgia, chronic gastritis, the passage of gall-stones, cirrhosis of the liver, and carcinoma of the stomach. The differentiation of the latter complaint will be given later. (a) In certain cases of cirrhosis of the liver hematemesis is met with, but here there is absence of all the other characteristic symptoms of ulcer, and the presence of a group of symptoms and physical signs pointing to disease of the liver. (b) Hepatic colic simulates, though not closely, ulcer of the stomach. The sudden onset, the longer duration of the attack of pain, its sudden complete cessation, the presence of jaundice and certain physical signs presented by the liver, suffice to distinguish this affection from gastric ulcer. (c) Chronic gastric catarrh with hematemesis simulates ulcer of the stomach in many particulars. The great diminution in the proportionate amount of hydrochloric acid found in chronic gastric catarrh and the increased amount in gastric ulcer are facts that will help materially in discriminating these two diseases. When they are associated with one another my observation teaches that there is an excess of HCl present; hence a proportionately diminished amount of HCl probably argues against the presence of ulcer. The vomiting in ulcer is combined with severe paroxysms of pain; not so in chronic gastritis, and the vomit in the former contains larger quantities of blood than in the latter disease. (d) Doubtless ulcer of the stomach has often been mistaken for neurotic gastralgia, and the discrimination cannot always be accomplished to a certainty. Their chief differential points may be conveniently arranged thus:

#### GASTRIC ULCER.

The paroxysms of pain usually come on at a definite period after eating.  
Eating rarely relieves pain.  
Tenderness on pressure over a certain limited area in the epigastrium.  
Pressure usually aggravates, and only occasionally relieves patient during paroxysm of pain—not during the intervals between seizures.  
In the intervals between the attacks gastric disturbances, more or less severe, are present; also tender point frequently.  
Hematemesis present in nearly one-half of the cases.  
General health often much impaired, particularly late in the affection.  
History of certain occupations, anemia, chlorosis, amenorrhea, tuberculosis, and diseases of the heart common.  
Most frequent from fifteen to thirty-five years of age.  
Physical signs of a mass may be present.  
Dilatation may coexist in the late stage.  
Hyperacidity of gastric juice usually present.  
Improvement follows rest and regulation of diet.

#### GASTRALGIA.

Paroxysms more frequent when the stomach is empty than soon after meals.  
Eating usually brings relief.  
Tender spot absent. General hyperesthesia of the skin often present.  
Pressure almost always relieves the pain.  
  
In the intervals between attacks no gastric disturbances present, as a rule.  
  
Hematemesis absent.  
General health less affected than in ulcer.  
History of neurasthenia, neuralgia, and hysteria common.  
Most frequent before or near the menopause (in the female).  
Signs of tumor always absent.  
Dilatation never present.  
Hyperacidity present only in certain forms (*supra*).  
Regulation of diet has no effect.



The **prognosis** is obviously uncertain. The average mortality is about 20 per cent. Such grave complications as free bleedings and peritonitis have been discussed sufficiently in the Clinical History. Among thoracic complications, pneumonia, tuberculosis, and left-sided perforative empyema are those most frequently encountered. They all render recovery almost positively hopeless. The possibility that the resulting scar may cause persistent gastralgia, and the probability that a cicatrix surrounding the whole or any part of the pylorus may cause obstruction at this orifice, followed by dilatation, must be kept in remembrance. Carcinoma may develop in the floor of an old ulcer in subjects who, on account of a predisposition, furnish a suitable soil.

**Treatment.**—The treatment of gastric ulcer embraces three leading objects: (1) Of paramount importance is *absolute rest for the stomach*. This is to be accomplished by maintaining the recumbent posture in bed, on the one hand, and by rectal feeding, wholly or partly, on the other. This mode of alimentation will be discussed presently. Perfect rest constitutes the best-known safeguard against those serious accidents that intervene suddenly in the course of this affection. It also ensures more rapid cicatrization than any other single agent. The process of repair is very slow under the most favorable circumstances; hence the patient should be informed at the outset that from four to six months, at least, must be spent in bed. (2) *The careful regulation of the diet*. It is not possible for the stomach, when the seat of ulcer, to digest the normal amount of nitrogenous food without being injuriously affected thereby. Those articles of diet should be employed that are digested and assimilated chiefly in the intestinal tract. But, though the patient is fed by the mouth, this should be supplemented by rectal feeding almost from the beginning. By pursuing this combined method and giving per rectum but a limited amount of albuminous food the vital forces can more effectually be supported. Failure to cure cases of gastric ulcer is often due to the fact that but little nourishment is supplied to the system, the patient's general strength being allowed to become exhausted quite early. Frequently the stomach is so irritable as to render it exceedingly difficult to introduce into it even a fractional part of the amount of food necessary to support life properly; and in all cases that I have seen the amount of food that could be taken by the mouth was really inadequate, considering the dietetic requirements of the disease. Should nutrient enemata not be well borne, they may be discontinued until the unpleasant symptoms have subsided, and then resumed immediately. By giving only a portion of the food in this manner rectal feeding may be continued for a long period without disagreeable intestinal symptoms. The following dietary will be found useful: At 7 A. M. give 100 c.cm. (3iij) of Leube's beef-solution; at 11 A. M., 200 c.cm. (3vj) of pancreatized milk-gruel;<sup>1</sup> at 3 P. M., 200 c.cm. (3vj) of peptonized milk or skimmed milk or buttermilk; and at 7 P. M., 200 c.cm. (3vj) of pancreatized milk-gruel; in addition, the following by rectal injection: at 8 A. M., 6 ounces of pancreatized milk-gruel, with  $\frac{1}{2}$  ounce of bovinin and 10 drops of tincture of opium, this to be repeated at 2 P. M. and 8 P. M. If the nutrient enemata must be discontinued for a time, the

<sup>1</sup> The milk-gruel is prepared with wheaten flour or arrowroot, mixed with an equal quantity of milk.

regular diet must be increased proportionately. If, on the other hand, the stomach rejects the above-mentioned food, then the feeding must be, for a time, exclusively rectal; this is quite practicable if the proper choice be made of nutrient preparations. In addition to the substances before mentioned we may employ from 4 to 6 ounces (150–200 c.cm.) of Leube's beef-solution, or the same amount of defibrinated blood or pancreatized milk with brandy. DaCosta reported recently a number of instances that were cured by a diet of ice-cream. It has been recommended to employ lavage when the stomach is exceedingly irritable, but the use of the stomach-tube is liable to damage the ulcer even in the most careful hands. The good effects from washing out the stomach for uncontrollable vomiting and pain have, however, been frequently witnessed. It may often be satisfactorily accomplished by the use, internally, of 1 pint ( $\frac{1}{2}$  liter) of warm water containing a few grains of sodium chlorid or bicarbonate, sipped slowly when the stomach is comparatively empty.

If at the expiration of two months the condition of the patient indicates that the reparative process is far advanced, then well-boiled rice, stale bread, and potatoes may be allowed; and later eggs, oysters, fish, and sago, the patient not being allowed to assume ordinary solid diet for at least six months. (3) *The medicinal treatment*, which is altogether subsidiary to the dietetic, has reference to two ends: (a) Promotion of the healing process. We cannot be certain that any remedial agents at our command can accomplish this object, and yet it is our duty to attempt it. Of the efficacy of alkaline remedies in this disease we are thoroughly convinced; in neutralizing the hyperacidity of the gastric secretions they fulfil an important indication, since the excess of HCl must have an unfavorable effect upon the ulcer. Of these, sodium bicarbonate (in full doses) or the alkaline purgative mineral waters, as Carlsbad, Kissingen, Hunyadi Janos, are most useful. The Carlsbad salts are also highly beneficial. They may be prepared artificially as follows: sodium sulphate, 50 parts; sodium bicarbonate, 6 parts; sodium chlorid; 3 parts—of which a teaspoonful may be taken in hot water, fasting, in the morning. The preparations of bismuth may be given in combination with antiseptics, which latter are especially to be recommended. Fleiner's method of giving 10 gm. of bismuth in 200 gm. of warm water on an empty stomach, and then allowing the patient to drink several swallows of water, and afterward placing him in the horizontal position with the hips elevated for about an hour, has yielded gratifying results. About 200 gm. of bismuth administered in the above manner usually suffice to effect a cure (Savelieff). Silver nitrate has long enjoyed an enviable reputation in this disease. For the chronic gastric catarrh which is very generally associated with ulcer, silver nitrate, as before stated, is most efficient, and may be combined with small doses of opium or hyoscyamus. The previous general condition of the patient is frequently unfavorable to the successful healing of the ulcers, and to combat the anemia and chlorosis that are often present we may employ iron and arsenic. The albuminate of iron has been warmly recommended, and small doses of Fowler's solution of arsenic are generally well borne by the stomach; the former may also be given hypodermically. When organic cardiac diseases are concom-

itants they should receive careful attention, and the recognition and treatment of all associated diseases must not be overlooked if the physician would obtain good results.

(b) The relief of urgent symptoms. The preceding measures relating to the diet and treatment, and particularly small doses of the extract of opium combined with silver nitrate, often relieve the pain. Mild counter-irritation is also of service, but warm poultices should not be employed if hematemesis be present in however slight a degree. The application of cold to the epigastrium in the form of an ice-bag sometimes alleviates the pain, though quite as often it fails to benefit. For the severe gastralgic attacks morphin administered hypodermically, at varying intervals, is demanded. Gerhardts prescribes three or four drops of liquor ferri chloridi in a wineglassful of water.

*Vomiting*, when not excessive, will be allayed by the use of the agents already mentioned, and bismuth, creasote, silver nitrate, and opium are especially useful; chipped ice, with a small amount of brandy thrown over it, is also of value. When obstinate the following remedies, in small doses, may be tried separately: cerium oxalate, potassium bromid, tincture of iodine, cocain, chloral, and hydrocyanic acid.

For the *hematemesis* the application of a broad, flat ice-bag, together with the use of ergot hypodermically, will usually suffice.

If the signs of *peritonitis* due to perforation should develop, the measures to be promptly instituted are—opium to relieve the intense pain, saline purgatives in small doses and at short intervals until free purgation results, and the flat ice-bag locally. It is important at the earliest moment to call a progressive surgeon to the case.

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## CARCINOMA OF THE STOMACH.

**Pathology.**—Next to the uterus, the stomach is the most favored seat of carcinoma. In a total of over 30,000 cases studied by Welch, 21.4 per cent. were found to show involvement of this organ. With reference to the parts of the organ most frequently attacked, Welch analyzed 1300 cases with the following results: pyloric region, 791; lesser curvature, 148; cardia, 104; posterior wall, 68; greater curvature, 34; anterior wall, 30; fundus, 19. The forms of gastric carcinoma noted are columnar epithelial (including colloid) and the glandular carcinomata (embracing encephaloid and scirrhus). The epitheliomata grow from the lining epithelium whilst the encephaloid and scirrhus are new growths from the glandular epithelium proper. The last two forms are therefore similar in structure, the differences possibly being due to variations in their growth (rapid in the encephaloid and slow in the scirrhus variety), and to the consequent varying proportion between fibrous tissue and cells; the encephaloid cancers are thus soft, and readily break down on their surface, forming large ulcers that have a clean floor, while the scirrhus cancers are hard and firm. Columnar epitheliomata are frequent, and are situated at the pyloric end of the stomach, where the glands are formed of a single layer of columnar cells on a basement mem-



brane. They are often the seat of colloid degeneration. Squamous epitheliomata occur only at the cardiac end, at the esophageal opening. All the varieties mentioned are prone to produce secondary new growths in adjacent organs, the scirrhus, however, manifesting a less marked tendency to metastasis than the others. They occur either as circumscribed tumors or as a diffuse infiltration, and in the immediate vicinity of the gastric carcinoma there is a marked thickening of the muscular coat and other tissue-elements.

**Etiology.**—The factors bearing upon the etiology of gastric carcinoma may all be regarded as **predisposing causes**. Of these *age* is the most potent. Of 2038 cases examined by Welch with reference to this point, 75 per cent. occurred between the fortieth and seventieth years, 24.5 per cent. between forty and fifty years, and 30.4 per cent. between fifty and sixty years. It is an exceedingly rare affection in very young persons. *Heredity* stands next to age as a causal factor, though it is far less influential. Welch analyzed 1744 cases, and found that a family history of carcinoma was present in about 14 per cent. *Sex* has little if any influence. The extent to which gastric carcinoma is dependent upon previous disease of the stomach is not definitely determined, but in persons that are predisposed to the affection by reason of age or heredity, the cicatrix of an old ulcer or a pre-existing chronic catarrh of the stomach may become additional causative factors of no mean importance. Strümpell has called renewed attention to the probable relation between gastric ulcer and gastric carcinoma, citing in confirmation the interesting discovery by Hauser of atypical growths of epithelium in the scars of gastric ulcers. Schmidt also has recently found the same cell-degenerations around both cancerous and ulcerous growths.

**Clinical History.**—Prior to the development of gastric carcinoma the symptoms of catarrhal dyspepsia may be present for a variable period of time. The onset is often, comparatively speaking, abrupt. It may, however, be insidious, and be marked more by the evidences of failing general health and strength than by distinct local subjective symptoms. *Anorexia* is commonly present, though occasionally the appetite remains unimpaired. A sense of oppression, rarely amounting to true cardialgia, and frequent eructations, come on soon after eating. In many cases but little *pain* is complained of, whilst in a lesser number pain is a prominent symptom throughout the entire course of the affection. Its character is very often described as lancinating, less often as burning or gnawing; the latter form of pain is due, most probably, to associated and secondary ulcers. The pain is often referred to the shoulders and the back or loins. *Vomiting* is infrequent, excepting in the more advanced stages of the disease, when it is almost constantly present to a greater or less degree. During the early stages it is due to the catarrhal irritation, later to obstruction. When the latter is at the cardiac orifice, the pain occurs at once after eating; when at the pylorus, it appears several hours after meals. Vomiting may also be caused by the occurrence of fermentation in large accumulations in the stomach. The *vomit* has few, if any, of the physical characteristics noted in simple ulcer of the stomach. Free hematemesis is very rare; when, however, the surface of the new growth ulcerates, there is almost invariably an occasional slow oozing of blood into the stomach. It is

here acted upon by the altered gastric juice, and the black hematin resulting from the transformation of the red hemoglobin gives rise to the well-known "coffee-ground" vomit of carcinoma of the stomach.<sup>1</sup> It is to be recollected, in this connection, that the chocolate-colored appearance of the vomitus is not found alone in carcinoma of the stomach, but may also occur in non-malignant disease and under other abnormal conditions of the gastric contents. The *chemical examination* of the gastric contents is of prime diagnostic importance, showing as it does the almost constant absence of free HCl. Riegel has recently given emphasis to the fact that the presence of free HCl, supposing the examinations to be properly made (by the use of the color test) and sufficiently often repeated, speaks almost positively against carcinoma. In not one of 154 artificial digestive experiments was albumin digested in this disease. Rare cases do, however, occur in which free HCl is present, as when carcinoma of the stomach is secondary to an ulcer, the free acid in these conditions being usually increased. More important than the latter small class of cases are those instances, not of carcinoma of the stomach, in which free HCl is absent; these are carcinoma of the esophagus, extensive amyloid disease, advanced cases of renal disease, and the febrile state. Riegel also noted the absence of free HCl in carcinoma of the duodenum.

Two leading views are held concerning the cause of the failure to find HCl: (1) That it is due to inflammatory degeneration of the mucous membrane, commencing as a catarrhal inflammation and advancing to interstitial change and atrophy (*Rosenheim's view*); (2) that the absence of the acid is due not so much to non-secretion as to its combination with some substance arising from the carcinoma (*Riegel's view*). *Lactic acid* occurs in the stomach in carcinoma more often than in any other condition, owing to the presence of the two essential conditions—an absence of the antifermentative (HCl) and the stagnation of food. The *microscopic appearances* of the vomitus are in some ways identical with those observed in gastric ulcer, and if it be examined speedily, red blood-corpuscles may rarely be seen. The microscope also, very occasionally, reveals pieces and bits of cancer-tissue, and Kaufmann, Hammerschlag, and others emphasize the frequency of long bacilli. It has been claimed that sarcinæ are present, but Oppler says that they never occur when HCl is present, and Riegel says that they are very infrequent. Chronic interstitial gastritis and atrophy of the mucosa may, among other conditions, show symptoms in addition to the absence of HCl.

**Physical examination** often discloses the presence of a tumor. *Inspection* may reveal an irregular tumor, particularly if the patient be much emaciated. When dilatation exists the outlines of the organ may be seen. On *palpation* the new growth, in a majority of cases, may be felt through the abdominal walls, though often not clearly, as a hard, nodular, and sometimes movable mass. Though this generally appears in the epigastrium, it must be recollected that it depends upon the part

<sup>1</sup> *Teichmann's test* for hematin crystals may be employed as follows: Place a drop of the "coffee-ground" material upon the slide and add a few crystals of sodium chlorid. Then introduce a few drops of acetic acid beneath the cover-glass, and on warming hematin crystals will form.

involved; also that a tumor united with the wall of the stomach frequently changes the position of the organ; hence the mass may be felt outside the normal boundaries of the stomach. The displacement is usually in the downward direction owing to dilatation, and the tumor may be found on a level with the umbilicus or even lower still. Less frequently, on account of its mobility, it is discovered in such unlooked-for situations as the right or left hypochondriac region. The varying degree of fulness of the stomach will obviously alter the position of the tumor. When situated at the cardia it is beyond reach of palpation; when attached to the lesser curvature of the stomach or the posterior wall, it is rarely to be felt unless it be of large size. The new growth cannot be definitely made out when it assumes the form of a diffuse infiltration, though it offers increased resistance to the palpating fingers and exhibits more or less tenderness on pressure. Usually the patient lies in the dorsal decubitus during the examination, with the limbs drawn up, being instructed at the same time to breathe regularly while opening the mouth. The detection of a tumor when in an unfavorable situation may be facilitated by shifting the patient's position from the dorsal to the lateral, the standing, or the knee-elbow position respectively; at the same time one or two tumblers of some carbonated water should be given, with a view to distending the stomach and bringing the tumor within reach. Pulsations are frequently communicated from the aorta to the palpating hand through the tumor. If the growth is situated at the lesser curvature, a deep inspiration will often cause the tumor to fall lower, and thus become accessible to palpation. *Percussion* over the seat of the new growth causes a muffled tympanitic resonance; superficial percussion, however, frequently gives absolute dulness.

The presence of *metastatic new growths* in the liver and enlargements of the supraclavicular or inguinal lymph-glands are of value in the diagnosis. In one instance that I saw in the Philadelphia Hospital a nodule the size of a walnut protruded from the umbilicus, leading to the suspicion that gastric carcinoma might be present, though the general symptoms pointed strongly to chronic gastric catarrh at the time. Subsequently, however, a round and somewhat nodulated mass, situated near the pyloric end of the stomach, could be readily grasped. In several instances in which the lymph-glands in the groins and the supraclavicular spaces were the seat of enlargement a probable diagnosis of abdominal carcinoma was made in the absence of positive symptoms and physical signs, and the diagnosis was borne out at the autopsies.

*General Symptoms.*—Quite early in the disease such evidences of general nutritional disturbance as loss of flesh and anemia may be observed, and, obviously, cases attended with constant anorexia and vomiting will earliest manifest the wasting processes. Almost from the beginning the face gradually assumes the cachectic appearance which, in the advanced stages, becomes so characteristic of gastric carcinoma. Anemia soon becomes a prominent feature. There is a waxy pallor of countenance, and the cerebral symptoms, as well as the peculiar cardiac murmurs of anemia appear. The blood frequently presents peculiarities that bear a resemblance to those seen in pernicious anemia, and sometimes there is a marked poikilocytosis. Welch has recorded an instance in which the ratio of white to red blood-corpuscles was 1 to 20, but the blood-count



and the estimation of hemoglobin rarely show the marked reduction in the number of corpuscles that is seen in progressive pernicious anemia; leukocytosis is also much more frequent. There is an almost constant absence of digestive leukocytosis in carcinoma; this, however, is a symptom possessing mere corroborative value, since it also often occurs in chronic gastritis and severe anemia. "When any degree of anemia is presented nucleated red corpuscles may be found in dry and stained specimens, and this method of examination may be of much service when an actual blood-count is impossible. The condition is, however, an anemia with wasting, and the layer of panniculus is not retained as in the ordinary forms of pernicious anemia" (Osler). The causes of the profound anemia met with in this affection are not quite plain, since frequently it becomes pronounced before the nutritional disturbances (shown by a loss of flesh) have become marked.<sup>5</sup> The fact that metastatic cancer has been found to be abundant in the marrow of the bones is significant in this connection, as pointing to the probable interference, in some instances, with the blood-producing function of the bone-marrow. In advanced cases moderate edema of the ankles and of the backs of the hands is frequently observed, and is probably dependent upon excessive anemia. The temperature at first shows no abnormalities, as a rule, though after the cachexia has become decided it is often subnormal. Sudden elevations of temperature (103° to 104° F.—40° C.), preceded by rigors and followed by profuse sweating, are rarely observed. The explanation of their occurrence is to be found in the fact that suppuration sometimes takes place in the bases of the cancerous ulcers. The mind almost invariably remains clear to the last, though delirium may occur near the close.

**Complications.**—*Intestinal symptoms* are frequently observed, and *constipation* in particular is quite common. It is apt to alternate with *diarrhea* toward the close of the disease, or diarrhea may in the later stages become a permanent and obstinate symptom. Some of the complicating conditions have reference to the *secondary* new growths. When, as frequently happens, the liver is implicated, *jaundice* is not uncommon, being associated with signs of hepatic enlargement. Indeed, so prominent may be the symptoms and physical signs referable to secondary carcinoma, of the liver as entirely to mask the more or less latent forms of carcinoma of the stomach. The mesenteric and retro-peritoneal lymph-glands or the lungs may be the seat of secondary carcinoma, though in these situations it rarely gives rise to characteristic symptoms. Occasionally the new growths spread to the peritoneum and sometimes give rise to ascites. As has been stated under Pathology, perforation may rarely occur, and we then have the pronounced and rapidly supervening symptoms of diffuse peritonitis. *Fistulous communications* between the stomach and the transverse colon or the small intestine—the latter rarely—may also occur. *Nervous symptoms* may be regarded as complicating conditions, and sometimes hasten the fatal termination; the patient becomes somnolent or, rarely, even comatose; the breathing is difficult and the respiration deep and labored. This mode of termination I noted in one of my own cases. Traces of *albumin*, and in the later stages tube-casts, may be present in the urine. An increased quantity of *indican* has frequently been noted; acetone and diacetic acid are present in rare instances.

**Atypical Forms.**—The disease rarely is entirely latent, and most often in persons previously much enfeebled and in the aged. In other instances the more prominent above-mentioned symptoms, and particularly the characteristic pain, are lacking; hence it frequently happens that the presence of cancerous tumors in the stomach is not suspected until accidentally discovered, the symptoms being attributed to less grave conditions. In still other instances the development of a pronounced anemia and of the cancerous cachexia alone furnish ground for suspicion; when these are associated with dyspeptic symptoms, however slight, they should lead to a careful physical examination of the stomach as well as a chemical examination of its contents.

**General Course and Duration.**—The course of gastric carcinoma is invariably toward a fatal issue, death usually taking place before the expiration of two years. The average duration of the disease probably does not exceed one year. According to my own observation, when it occurs in emaciated persons it pursues a slower course than when occurring in apparently robust and fleshy individuals. No case of recovery from carcinoma of the stomach is recorded.

**Diagnosis.**—A positive diagnosis of gastric carcinoma is easily made when a tumor is demonstrable. It is, however, possible to diagnose the disease in the absence of a palpable new growth. The history, the presence of such characteristic symptoms as pain, coffee-ground vomit, the existence of dilatation of stomach, the constant absence of free hydrochloric acid, the constant presence of lactic acid after the Boas test-meal,—all occurring in an elderly person, together with marked anemia and emaciation, are sufficient to warrant a diagnosis. It is needful, however, to exclude the diseases other than gastric carcinoma in which the absence of free hydrochloric acid has been noted. If these points be carefully considered, there are few instances in which the diagnosis cannot be made with reasonable certainty. Of course all corroborative evidence must also be taken into account.

**Differential Diagnosis.**—A gastric carcinoma presenting a discernible mass is liable to be mistaken for a *cicatrizated ulcer*, for *carcinoma of the pancreas*, of the *transverse colon*, *duodenum*, *omentum*, and the *left lobe of the liver*, as well as for *aneurysm of the abdominal aorta*. The *aneurysmal tumor*, however, is smooth, and is not nodular like the cancerous growth; moreover, it gives rise to an expansile impulse, and not to the heaving impulse of a solid growth. In aneurysm the characteristic gastric symptoms, the peculiar cachexia, emaciation, and marked anemia that belong to carcinoma are wanting. In *pancreatic carcinoma* the tumor is always fixed; there is an absence of the coffee-ground appearance of the vomit and of dilatation, and free HCl is present in the gastric contents. Further than this, fat may be present in the stools and sugar and fat in the urine. *Carcinoma of the transverse colon and omentum* will be excluded by the presence of such significant symptoms as gastric hemorrhage and the consequent hematemesis, by a chocolate-colored appearance of the vomitus, and the permanent absence of HCl. In this connection two facts should be re-stated: (1) That in carcinoma of the duodenum free HCl has been found to be absent from the gastric contents; (2) that in carcinoma following gastric ulcer free HCl may rarely be present. The points of difference between carcinoma of the

stomach and of the left lobe of the liver will be referred to under the latter head.

*Simple round ulcer* of the stomach may in cicatrizing give rise to a small tumor, followed by pyloric stenosis and secondary dilatation—an exact counterpart of the course of gastric carcinoma. Under such circumstances the clinical picture is at first apparently identical with that of pyloric carcinoma, though it presents a few distinguishing features. Great reliance should be placed on the age of the patient, the presence of HCl in the gastric secretions, the grave gastric disturbances with hematemesis, together with the longer duration of ulcer (more than two or three years), which is almost pathognomonic of the latter disease.

Simple gastric ulcer and chronic gastritis are often confounded with carcinoma of the stomach. The facts of greatest value in the discrimination of these three affections are so well presented by DaCosta that they are, in the main, here subjoined :

CHRONIC GASTRITIS.	GASTRIC ULCER.	GASTRIC CARCINOMA.
Not confined to any age. More common in middle-aged or elderly people.	May occur in middle-aged persons, but is most frequent in young adults, especially women.	Most common in elderly people; rarely occurs in persons under forty years of age.
Pain at the epigastrium somewhat augmented by food; soreness is also present. Both are constant, although comparatively slight.	Pain at the epigastrium much augmented by food; subsides when this is digested: paroxysms of pain, not lancinating; strictly localized soreness to touch in epigastrium; sometimes a painful spot over lower dorsal vertebræ. Intermissions in the pain of considerable length are frequent.	Pain frequently of a radiating kind, often paroxysmal, not infrequently severe and lancinating, but not of necessity associated with soreness. Little or not at all affected by food. Pain rarely remits; never intermits for any considerable time.
Symptoms of indigestion marked.	Symptoms of indigestion sometimes very slight.	Symptoms of indigestion marked. Anorexia; extreme acidity of stomach.
Sometimes vomiting.	Vomiting may be present or absent.	Vomiting a very frequent symptom.
No hemorrhage, or but trifling hemorrhage; at most blood-streaks in vomited matter.	Abundant hemorrhage from the stomach common.	Hemorrhage not very abundant, but frequently occasioning coffee-ground-looking vomit.
Bowels constipated.	Bowels may or may not be constipated; usually are.	Bowels obstinately constipated.
No fever.	No fever.	Intercurrent attacks of slight fever may occur; but temperature often subnormal.
Not much emaciation; no cachectic appearance.	Frequently extreme pallor and debility.	Gradual and progressive loss of flesh, and debility; and at times, with the cachexia, hypertrophy of the peripheral lymphatic glands, especially above the clavicles.
Disease may be relieved or cured; is often of very long duration.	Duration uncertain; may get well, may run on rapidly to perforation; on the other hand, may last for years.	Average duration one year; may be shorter, but seldom longer.



No tumor.	Rarely a tumor.	Generally a tumor.
Contents of stomach almost always contain free hydrochloric acid.	Hydrochloric acid in excess in contents of stomach.	No hydrochloric acid in contents of stomach.
No dropsy.	No dropsy.	Edema of ankles often met with.
No lactic or fatty acids after the rigid Boas test-meal.	No lactic or fatty acids after the rigid Boas test-meal.	Lactic acid present after Boas's test-meal.

**Treatment.**—The diet should receive careful attention, and it will be necessary to adapt it to the peculiarities of the individual case. In general terms, articles of food that are digested and assimilated in the intestines should be employed. After well-marked evidences of pyloric obstruction appear we may add greatly to the comfort of the patient by limiting the dietary to liquids, and by predigesting them if they are not otherwise well borne. Should the stomach reject all food, rectal alimentation should be promptly instituted. The medicinal treatment of gastric carcinoma is altogether symptomatic, no remedy with any power over the lesion having been found. The more troublesome symptoms—namely, pain, vomiting, and constipation—are to be met on general principles. Should free hematemesis occur, it should be treated as previously indicated under Gastric Ulcer. The claims that have been advanced in favor of arsenic and other preparations as possessing power to control the progress of gastric carcinoma have not been confirmed by any extended experience. If dilatation coexists, it is to be managed in accordance with the recommendations found under that heading. Surgical treatment may also be called into service.

## HEMATEMESIS.

HEMATEMESIS is a symptom that occurs in widely different diseases and conditions. Inasmuch as it is due to various causes in diseases other than those of the stomach, it is hardly to be properly classed among gastric affections, and, at all events, is not entitled to more than a brief separate description.

**Etiology.**—Among the causes of hematemesis are—1. Injury to the stomach; 2. Diseases of its coats (carcinoma and ulcer); 3. A mechanical impediment to the portal circulation; 4. Vicarious menstruation; 5. Alterations in the blood; 6. A disease of some neighboring organ, such as carcinoma of the pancreas, may perforate the gastric coats and open its vessels.

**Symptoms and Diagnosis.**—If the fact that it is always a symptom, and not the disease itself, be recollected, the importance of recognizing its special causal condition in each instance will be obvious. The manner of its occurrence and the characteristics presented by the blood often give a clue to its nature and origin. Thus, we have seen that the clinical signs in hematemesis due to carcinoma and ulcer of the stomach vary greatly, being almost peculiar to each. This fact must, however, be weighed with the history and symptoms of the case in which it may occur; in this manner, and in this manner only, can errors be avoided. A process of exclusion is the best way to reach a decision. If a careful inquiry determines the absence of morbid lesions of the stomach, such as

carcinoma, ulcer, or chronic gastritis, then the other organs of the abdomen, and more particularly the liver, must be examined. If this and the heart be found to be healthy, attention should then be turned toward the state of the blood, since the presence of any specific fever may readily account for the hematemesis. Should the blood present nothing abnormal, it may be found that the menstrual or other habitual discharge has become suppressed.

**Differential Diagnosis.**—It is to be recollected that the source of the blood may be other than the stomach. Rarely, an abdominal aneurysm bursts into the stomach; occasionally, too, a thoracic aneurysm opens into the esophagus, whence the blood speedily finds its way into the stomach. A careful consideration of the history and of the attending symptoms, together with a thorough physical examination, will, after excluding the various conditions causing true gastric hemorrhage, lead to a correct interpretation of the phenomena. Blood coming from the throat, tonsils, mouth, or the respiratory organs, including the nose, is sometimes swallowed, and afterward ejected by vomiting. To discriminate from this condition it is only necessary to make an examination of the lungs and elicit most carefully the history. It must also be recollected that hysterical females and malingerers have been known to swallow the blood of animals and other dark fluids, and vomit them subsequently. The vomitus may resemble dark blood in appearance when stained by bile or iron or after a free indulgence in wine. The diagnosis between hematemesis and hemoptysis is sometimes attended with difficulty, and the points of contrast have therefore been placed side by side in the following table:

## HEMATEMESIS.

The history points to gastric, splenic, hepatic, or cardiac disease.  
A feeling of uneasiness, and sometimes of nausea or faintness, precedes the hemorrhage.  
The blood is ejected by vomiting; violent vomiting may excite cough.  
The blood is either clotted or fluid and dark; it may be mingled with remnants of food, and is acid in reaction.

## HEMOPTYSIS.

History of cough and other symptoms points to pulmonary or cardiac disease.  
A feeling of weight and uneasiness in the chest, a saline taste, and a tickling in the throat precede the hemorrhage.  
The blood is raised by coughing, though, if it be swallowed, vomiting may follow.  
The blood is bright-red, frothy, in small coagula, and alkaline in reaction.

**Prognosis.**—Hematemesis, except it be due to rupture of an aneurysm, rarely presents a hopeless prognosis. In cases of splenic enlargement, hepatic cirrhosis, or gastric ulcer it may prove fatal, either as the direct consequence of loss of blood or more gradually as the result of anemia and debility, induced by the bleeding.

The **treatment** has been detailed in the discussion of Gastric Ulcer.

## NEUROSES OF THE STOMACH.

## NERVOUS DYSPEPSIA.

**Definition.**—A functional disorder of the stomach, usually characterized by regularly (and sometimes irregularly) recurring attacks of gastric disturbance, followed by almost complete freedom from symptoms. The explanation of the symptoms is found in the well-known effect of

certain nervous influences upon the digestive function, and the term nervous dyspepsia embraces all the forms of gastric neuroses to be hereafter described. There are no local lesions detectable.

**Etiology.**—The vast majority of cases occur in highly emotional and hysteric persons, under such exciting conditions as great anxiety, violent passion, anticipation of pleasure; in short, any startling news or sudden excitement may cause it. The condition is most commonly met with in healthy-looking, ruddy-cheeked subjects, though it may also occur in the weak and pale-faced. The symptoms follow immediately upon the action of the exciting cause. A small percentage of instances are due to hypochondriasis.

**Symptoms.**—In the ordinary form the gastric secretions are normal, and the stomach is found empty after a test-meal within the physiologic time-limit. There is anorexia, which occasionally alternates with a voracious appetite. After meals the patient complains of distress and oppression in the epigastrium; eructations, and an occasional regurgitation of the acid liquid or solid contents of the stomach, with heartburn, will also be noted. Vomiting is not rare, and occurs independently both of the time of eating and of the character of the food. *Gastric peristalsis* is sometimes so well marked as to be readily felt and even visible through the stomach-wall. Kussmaul has called special attention to this symptom, which, I believe, belongs largely to nervous dyspepsia, though I shall refer to it separately under the designation of *peristaltic unrest* (*vide* p. 784). In every instance it is dependent upon the excitement of sensibility. The increased peristaltic waves, especially under emotion, excite cooing, gurgling sounds that are a source of great annoyance to the patient. Peristalsis, in which the movement occurs from right to left, has also been observed, and under these circumstances fecal vomiting may occur.

The *physical examination* sometimes reveals abdominal distention and hyperesthesia of the surface, but no localized tenderness, pressure with the broad hand usually affording relief from pain. *Nervous phenomena* always exist, and their correct interpretation is of the utmost importance in the diagnosis. The presence of headache, vertigo, numbness, should be noted, and also a coolness of the extremities during the exacerbations to which the disease is liable. The mental condition is unstable and illy regulated, and this fact furnishes a satisfactory explanation of the operation of the etiologic factors. The general health is in many instances not noticeably impaired; in others, particularly in those subject to frequent vomiting and complete anorexia, the general nutrition suffers considerably.

**Complications.**—The bowels are often constipated, are apt to be distended with gas, and may be the seat of an abnormal peristalsis that is transmitted directly from the stomach.

*Nervous dyspepsia with hypochondriasis* forms a group of cases in which the hypochondriasis may sustain a causal relation; it may be secondary to the gastric disturbances, however. In either event it is apt to become pronounced after the gastric symptoms have lasted a long time. The symptoms other than the nervous phenomena are quite similar to those previously described.

**Diagnosis.**—The course of nervous dyspepsia, in all of its clinical varieties, is chronic, and it not infrequently terminates in chronic catarrh of the stomach. The diagnosis is based on the following points: (a) The



etiologic factors. Here it is important to ascertain the particular mental influence that produces the gastric symptoms, taking also into consideration any well-recognized predisposing causes. (*b*) The course of the complaint and the absence of some of the physical signs and symptoms that would point positively to anatomic lesions of the stomach. When there is a catarrhal process, the symptoms become more pronounced immediately after taking food; this is not so, however, in the disease under consideration. The influence of the ingestion of indigestible substances upon sympathetic dyspepsia is often to relieve, or is of neutral effect, whereas in catarrhal indigestion it decidedly aggravates the condition. The dull pain after eating and the tenderness on pressure are usually more marked in the catarrhal variety than in the nervous type. The symptoms of the latter intermit from time to time, while they are more constantly present in chronic catarrh. The analysis of the contents of the stomach by means of the stomach-tube will also assist in the diagnosis. The gastric contents in cases of nervous dyspepsia are usually about normal, though any abnormality (even to complete achlorhydria) may occasionally be present.

**Prognosis.**—If there be an absence of any inherited predisposition, and if the cause is removable, complete recovery may be prognosticated. In a neurotic constitution, however, the tendency to recurrence, even after a decided improvement has taken place, is very strong. The most unpromising cases are those in which the causative mental influences are irremovable, though as to life the prognosis is not unfavorable. The patient himself is always of the opinion that he is suffering from a serious and incurable affection.

**Treatment.**—Every causal factor must be recognized and removed if possible. If we fail to accomplish this end, our efforts at cure will be unsuccessful. The dietary should be generous and composed of highly nutritious articles of food, and to convince the patient that his stomach is capable of digesting a full meal is the first duty of the physician, though the task is confessedly difficult. So soon as the patient realizes the truth in reference to his digestive capacity his sufferings are largely at an end. It is the nervous system that demands especial attention, and the internal treatment of the stomach is merely placeboic. Nerve-tonics combined with nerve-stimulants are often serviceable, and the following prescription will be found to be adapted to a certain proportion of cases:

Ry. Quininæ valerianat.,  
 Zinci valerianat.,  
 Ext. sumbul, aa. gr. xxx (2.0);  
 Strychninæ sulphat., gr. j (0.0648).

M. et ft. pil. No. xxx.

Sig. One after each meal.

Should the patient be anemic, iron and arsenic should be added to the above pill. A change of air from the city to the country, the mountains, or the sea-coast is usually followed by improvement. In some manner the patient must be extricated from the old surroundings under the influence of which the disease was started and has continued. Sea air has seemed to me to be more serviceable than mountain air in these cases, though I believe it to be an axiom in climatic therapeutics that the latter confers more lasting benefits than the former. These patients are

often averse to taking exercise, but so great is the value of this sanitary measure that it should never be overlooked. Walking and the lighter gymnastics are especially useful. Cold sponging of the surface, followed by friction to the skin, should be practised daily for its effect upon the skin-circulation and the nervous system. Occasional lavage, hot and cold douches, electricity (intra- and extra-gastric), and gastric massage, may all be tried, and may prove of distinct advantage. In highly neurotic and hysteric females, as well as in those in whom nervous vomiting is a prominent symptom, the S. Weir Mitchell treatment is often attended with good results. The hypochondriac form is often intractable. Strychnin, however, if perseveringly used, and if coupled with a change of air, often proves beneficial. One of the most obstinate examples of this nature that I have ever seen occurred in a retired merchant living in Philadelphia. This man was finally cured in consequence of his own suggestion, resulting in his removal to the country and engaging in farming on a small scale.

### NEUROSES OF SECRETION.

#### HYPERCHLORHYDRIA.

(*Hyperacidity.*)

**Definition.**—An augmentation of the secretory function of the stomach during the digestive period, resulting in the presence of an excessive amount of hydrochloric acid.

**Etiology.**—Hyperacidity is common during digestion, and its cessation in most instances is due to some one of the psychologic influences mentioned under Nervous Dyspepsia (grief, great anxiety). Less frequently, it is induced by mental over-taxation, and it is often met with among the professional classes. Highly-seasoned foods and alcoholic intoxicants may sometimes occasion the condition.

**Symptoms.**—Hyperchlorhydria may be *continuous*, though more often it is *discontinuous* and lasts from a few hours to several days. After the periodic form has lasted a long time it may gradually become a permanent condition. The patient at first complains of *uneasiness* in the epigastrium one or two hours after meals. Later, this amounts to *pain* of moderate intensity, and soon follows every meal after a like interval. The duration of the pain is from one to three hours. Acid eructations are frequently noted. The increase of hydrochloric acid interferes with the digestion of starches, and thus tends to increase the pain. On the other hand, however, a diet composed of albuminoids often affords relief, and the salts of the alkalies also ease the pain. Associated *nervous symptoms* (headache, dizziness) are often observed, though the bodily nutrition is usually well maintained. *Palpation* of the epigastrium may show a diffused tenderness. Evidences of moderate dilatation of the stomach sometimes appear, and splashing sounds may be detectable.

**Diagnosis.**—Though the diagnosis of hyperacidity is made probable by the above-mentioned symptoms, it is rendered certain only by a repeated analysis of the gastric contents. The findings, according to Einhorn, are—(1) On examination of the stomach in the fasting condition, the organ either is found empty or contains only a few cubic centimeters of juice; (2) one hour after Ewald's test-breakfast the hyperacidity is greatly increased, owing to the great amount of free HCl.

**Gastric ulcer** must be eliminated. In this disease the pain is aggravated immediately after eating, and is not relieved by albuminous food, nor by large doses of alkalies as in hyperchlorhydria. In ulcer, moreover, the pain often leads to vomiting, and severe, painful attacks frequently occur at night.

**Gastro-succorrhœa** (*Reichmann*); **Gastroxynsis** (*Rossbach*).—In this affection there is an increase of hydrochloric acid, either constantly or intermittently, when no food is present. An *epigastric gnawing pain* and nausea appear in the full bloom of health. The nausea soon results in the *vomiting* of enormous quantities of gastric contents. The appetite is lost, but the thirst is excessive, and the amount of drink taken and of liquid vomited are proportional. During the night or in the early morning hours the patient commonly vomits large amounts of a clear or bile-tinted liquid containing hydrochloric acid and the gastric ferments in excess. This may be followed by persistent vomiting, attended with much retching. After a lapse of a few hours the ejection of a large quantity of highly acid liquid may be repeated. The pain often becomes intense, headache is common, and a tendency to collapse is usually marked. The attacks last, as a rule, about two or three days, when they quite abruptly give place to apparent good health. *Recurrence* at the end of periods ranging from a few months to a year or more are common.

The *diagnosis* is made upon the presence of the clinical symptoms and course, as well as upon the results of oft-repeated analyses of the vomitus. Gastric ulcer and certain organic spinal and cerebral nervous affections, in which there is excessive gastric secretion, must be excluded before an absolute diagnosis can be made.

**Gastro-succorrhœa Continua Chronica.**—Reichmann first described a condition characterized by a *constant secretion of gastric juice* either in the absence or presence of food. The symptoms are much the same as those in hyperacidity, but tend to become continuous, so that the vomiting finally becomes a daily occurrence. In the fasting state a highly acid secretion that contains no food-particles flows through the tube from the stomach. Albuminoids are rapidly and starches slowly digested by these patients, as is shown by an examination of the gastric contents three or four hours after the Leube-Riegel's test-meal (one plate of soup—400 c.c.—a large portion of meat, some potatoes, and a roll). The disease is quite rare, and must not be confounded with the organic diseases to which continuous gastric succorrhœa may be secondary and upon which it is dependent. Indeed, Schreiber, Boas, and others believe that this is almost always a symptom of gastric atony or gastric ulcer.

Leube has described a neurosis in which there is a constant **sub-acidity of the secretion**. During the digestive process the percentage of hydrochloric acid is *low*. I believe this condition to be rare as a pure neurosis; it is, however, of frequent occurrence as a symptom of such organic gastric affections as chronic catarrh and the like.

The **prognosis** in the foregoing affections is not bad as to life, and not infrequently a cure, even, can be effected.

**Treatment.**—The dietary embraces only nitrogenous articles of food, while the medicinal treatment should, in addition to meeting the general



neurotic condition, consist of full doses of sodium bicarbonate. Lavage daily, before the chief meal, is also sometimes beneficial.

### NEUROSES OF MOTILITY.

#### INCREASED PERISTALSIS OF THE STOMACH.

Gastric peristalsis is increased in various conditions, which will be considered *seriatim*, though briefly.

(a) **Belching and Eructations.**—These may be of *nervous origin* and are met with generally in *hysterical* subjects, and less frequently in *neurasthenics*. The air is swallowed, and then expelled with more or less noise, owing to an increased contractility of the stomach. The gas is *odorless*, and differs in this point from the gases of fermentative dyspepsia. Epigastric distress and distention often arise, and certain nervous phenomena, as anxiety or palpitation, may coexist. It must not be forgotten, moreover, that in hysterical subjects the belching may be from the esophagus alone.

(b) **Pyrosis** means regurgitation of the acid contents of the stomach into the esophagus and mouth, causing intense *burning sensations*. The stomach-contents are not necessarily hyperacid.

(c) **Rumination (*Merycism*).**—A rare affection in which the food is regurgitated into the mouth, the cud chewed, and again swallowed after the fashion of ruminants.

(d) **Nervous Vomiting.**—This is a *reflex neurosis* that may affect persons of any age, though most frequently it is seen in adult females with an hysterical tendency. Without previous nausea, and independently of the character of the food taken, the contents of the stomach are readily expelled or, more correctly speaking, regurgitated into the mouth, and then expectorated. Though this usually takes place after meals, it may occur without reference to meal-time—a feature that indicates its nervous origin. The *attacks* of vomiting are separated by longer or shorter intervals of excellent health. Periodic vomiting may also occur independently of hysteria or other nervous affections, as pointed out by Leube. The *course* is rarely unfavorable, though exceptional instances have proved fatal.

(e) **Peristaltic unrest (Kussmaul), or spasm of the stomach**, has been referred to under Nervous Dyspepsia. It has also been observed in compensatory hypertrophy of the stomach-wall following pyloric stricture. In a case of gastric carcinoma in my own care the supermotility of the stomach caused an almost immediate expulsion of the gastric contents, and even of the rigid test-meal at certain times.

**Treatment.**—To the regimenal management, including a hygienic mode of living, the attention of the physician should be primarily directed. The medicinal treatment is to be aimed at the causal or primary nervous affection. The valerianates and the bromids (the latter continued over a period of two or three months) often do good service.

#### DIMINISHED PERISTALSIS OF THE STOMACH.

(*Atony.*)

(a) **Pyloric Relaxation or Incompetency.**—This is a rare neurosis that allows the partially digested gastric contents to pass the portals of the

stomach prematurely. It likewise permits the regurgitation of the contents of the duodenum into the stomach. Its recognition is possible upon inflating the stomach, when gas may be seen to pass into the intestines, and also (even with greater certainty) upon the regurgitation of intestinal contents into the stomach.

(b) **Cardiac Relaxation.**—This condition leads to eructations and regurgitations, and when these are of aggravated form they impair the general nutrition. Ordinarily this state of affairs runs for years without marked ill-effects.

(c) **Atonic Dyspepsia** (*Atony*).—This may occur as a neurosis, though oftener it is secondary to chronic gastritis. It implies *hypomotility* or insufficiency. The chyme is retained in the stomach beyond the natural time-limit. There is an epigastric oppression with a distention of the organ during digestion that tends to become permanent. There are eructations of gas, an impaired appetite, and often constipation. The stomach is found empty in the morning, and six or seven hours after Leube's test-meal it contains some chyme. In the absence of pyloric stricture the hypomotility may be shown by the administration of salol (see Methods of Diagnosis).

**Treatment.**—The diet is to be regulated as in chronic gastric catarrh. It is rarely necessary to restrict the solids to the same extent as in the latter affection, but the quantity of fluids should be lessened. The patient must be taught to eat slowly and masticate thoroughly. His hygienic standard of living must be high, and he must not be allowed to over-use his mental faculties. Exercise in the open air and cold baths, properly regulated, are potent for good. Of medicines, strychnin stands first, and I have found the following formula of great service:

Ry. Tr. nucis vomicæ, f3ijss (10.0);  
 Inf. cascarillæ, q. s. ad f3iv (128.0).—M.  
 Sig. 3ij (8.0) three times daily.

Electricity is also indicated, and it is in these cases that intragastric faradization has given excellent results. The constipation is to be overcome by an appropriate dietary (green vegetables, Graham bread, an abundance of fruit). The fluid extract of cascara sagrada may be employed if dietetic means fail.

## NEUROSES OF SENSATION.

## CARDIALGIA.

(*Gastralgia*; *Gastrodynia*.)

**Definition.**—Severe paroxysmal pain in the epigastrium in the absence of gastric lesions. There are two other forms of this disease that are clinically identical with gastralgia, the one occurring in ulcer and carcinoma of the stomach, and the other in certain chronic nervous diseases, forming the so-called gastric crises, which will be considered hereafter.

**Etiology.**—There may be a history of an inherited predisposition to neuroses of various sorts. Such conditions as anemia, exhaustion from

repeated hemorrhages, excessive venery, and lack of nourishment also predispose to this affection. The female sex is more liable than the male, and in the former it appears to be dependent upon disturbances of the menstrual function or quite frequently upon hysteric conditions. It is sometimes excited, in those predisposed, by reflex irritation, by deep grief, worry, and great anxiety. Hypochondriasis and hyperacidity are also among its frequent causes.

**Symptoms.**—These are *sudden in their onset* as a rule, and quite characteristic. Occasionally the attack is preceded by anorexia, or it may begin with a sense of oppression and distention in the epigastrium, lasting for a few minutes. In any event, the onset of the attack proper is marked by *agonizing pains* in the epigastrium, that dart through to the back, and at times also pass around the lower ribs. The seizure lasts from a few minutes to an hour or two, and terminates with *eructations of gas*, or, less frequently, with vomiting. From the nature of the causative factors it is obvious that the gastralgie seizures are in no wise dependent upon the character of the food taken; hence the fact that they occur more frequently when the stomach is empty need occasion no surprise. Firm pressure over the epigastrium relieves the pain. *Nervous phenomena*, varying with the etiology of individual cases, are constant attendants, but cannot be detailed here. A distinct clinical variety is found associated with that form of nervous dyspepsia in which an excess of acid is secreted (*vide* Hypersecretion); this occurs at varying intervals. Many purely functional nervous disturbances are thus subject to the law of periodicity. I believe that a very small percentage of cases are caused by malaria, since I have met with two such cases in a distinctly malarial district, both of which yielded readily to quinin. The disease took on a desultory, periodic character, and was associated with other malarial symptoms.

**Diagnosis.**—The history, together with the characteristic symptoms of the gastralgie attacks and their time of occurrence, and also the absence of any local causes, will render a positive diagnosis easy in most instances. To discriminate this condition from *gastric ulcer* is sometimes difficult, but stress has been laid upon the differential points in the description of the latter condition.

**Prognosis.**—This depends entirely upon the causal condition. The disease itself has no intrinsic fatal tendency.

**Treatment.**—This is to be subdivided into (*a*) the treatment of the attack; (*b*) the management of the intervals between the seizures. The pain is, as a rule, sufficiently intense to demand morphin, which is best administered hypodermically in combination with atropin. This should not, however, be repeated unless urgently needed. In mild attacks the constant or the faradic current often affords prompt relief. Under these circumstances counter-irritation, together with the use internally of Hoffman's anodyne or chloroform in small doses oft repeated, sometimes suffices to relieve the pain.

(*b*) *The Management of the Intervals.*—Here the physician's efforts should be directed to the detection of the causes and their removal by appropriate means. In hysteric females I have obtained good results from the prolonged use of the valerianates, combining with them iron and arsenic, thus:



R̄. Zinci valerianat.,	gr. xvij (1.16);
Quininæ valerianat.,	gr. xxvij (1.74);
Ferri arseniat.,	gr. ij (0.129).

M. et ft. pil. No. xvijj.  
Sig. One after each meal.

A change of air is often highly serviceable, and should be advised whenever financial considerations permit. These patients are constantly in a more or less exhausted, anemic, and run-down condition, and a tonic plan of treatment is always indicated. The return of the attacks in malarial gastralgia may be prevented by the timely use of quinin and arsenic. In the intervals between the attacks digestion, as before stated, proceeds normally, and the stomach therefore requires no treatment. Constipation, if present, is a condition demanding relief, not, however, by the use of purgatives, but by such means as massage, a suitable diet, enemata, or laxative suppositories. The physician must so regulate the sanitary particulars of the patient's daily life as to put him in the best possible condition to improve the general nutritive processes.

#### HYPERESTHESIA OF THE STOMACH.

This is met with in functional and organic diseases, as well as in chronic gastric catarrh and other affections of the stomach. Again, it may occur as a neurosis, most frequently in chlorotic girls and women. There is an increased gastric sensibility, so that the mildest irritant produces *painful sensations* that may be either gnawing or burning in character. A feeling of fulness and nausea are among the common features of the complaint. Food and certain articles that are not easily digestible may afford relief, and, oppositely, fasting or restriction of diet may aggravate the condition. The complaint, however, is often aggravated during digestion, particularly after excessive indulgence in certain kinds of food (crabs, lobsters, oysters, strawberries). Cutaneous symptoms, as erythema and urticaria, may appear. *Hypochondriasis* is often associated. The above symptoms are dependent upon an individual idiosyncrasy.

**Treatment.**—At first a restriction of the diet to soft and liquid articles should be tried, and later a cautious return to solid food is to be made. Of medicaments, the bromids, given for a period of two or three months, have given the best results in my own hands. For the chlorotic type iron in the form of Bland's pill, in ascending doses, is the best treatment.

#### ANOREXIA.

This consists merely in a loss of appetite, and occurs in many organic gastric disorders. It may also be a primary gastric neurosis, the latter being often associated with gastric hyperesthesia. Anorexia sometimes leads to a repugnance to food and a degree of abstinence that may induce grave nutritional disturbance. Among exciting causes mental shock of any sort ranks first. In other instances the patient may experience hunger, but on attempting to eat *anorexia* quickly develops. The recognition of anorexia as a primary neurosis of the stomach is difficult in the extreme after the general nutrition has become seriously impaired.

Chronic dyspepsia, phthisis, and other diseases associated with emaciation and debility must be excluded before the diagnosis is established.

#### HYPEROREXIA.

(*Excessive Appetite.*)

This may either be symptomatic of other affections (*e. g.* diabetes mellitus) or it may be of nervous origin. It may also be paroxysmal (bulimia). The patient complains of burning sensations in the epigastric region and of an insatiable hunger. The symptoms of neurasthenia and hysteria are often in association. The local and general symptoms are relieved by food. Hyperorexia may become permanent (polyphagia) and induce great debility. It may also accompany other nervous disorders, as affections of the brain, exophthalmos, and migraine.

**Pica** is the term applied to the craving for substances not used as food (slate-pencils, dirt, chalk).

**Malacia** represents the desire for highly spiced dishes (mustard, salads, pickles, fruits).

The above conditions are met with in neurasthenia, chronic gastric affections, and chlorosis.

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## VIII. DISEASES OF THE INTESTINES.

### METHODS OF DIAGNOSIS.

**Examination of the Feces.**—Although the results are in most cases unsatisfactory, an examination of the feces should not be neglected, especially in the more serious affections of the intestine. This embraces—(*a*) a macroscopic; (*b*) a microscopic; (*c*) a chemical; and (*d*) a bacteriologic examination.

(*a*) The *macroscopic* appearances often suffice. A thorough inspection of the stools, a matter too often omitted, furnishes valuable points in regard to the presence or absence of coarse parasites, fragments of tumor, foreign bodies, concretions, blood, bile, pigment, fat, pus, mucus, undigested meat, and the like.

The shape, color, and consistence of the stools must be noted, and it is to be remembered that in these particulars, as well as regards their frequency, they exhibit a considerable range of normal variations, according to individual peculiarities, the character of food taken, and so on. It is to be recollected that normal stools contain fat in varying amounts, for the reason that only a limited quantity can be emulsified and taken up from the intestine. The naked eye may, at times, detect its presence from the "peculiar silvery appearance" of the feces. Fat in the stools (*steatorrhea*) is often pathologic, and the separate affections in which it is met with will be considered hereafter. The dejecta present a shining, tallowy appearance, either throughout or in circumscribed spots. Again, the fat may occur in the form of oil floating on the surface of liquid stools. Mucus is also visible, either as slimy or jelly-like masses, or as shreds and granules (sago-grains). Diarrheal

stools should be examined macroscopically with great care, and the same may be said of constipational dejections. The latter often assume a rounded form (*sheep's dung*) on account of their delay in the large bowel. They may attain to the size of a small orange, and may be, though rarely, enveloped in mucus or blood-streaked. Their color is dark. On the other hand, the stools may be colorless in cases in which the bile-ducts are occluded; these usually contain a large proportion of fat, though not invariably. The effect of certain drugs upon the color of the stools is to be borne in mind. When blood is intimately mingled with the feces, they have a reddish, dark- or blackish-brown (tarry) color, according to the quantity and the time allowed for decomposition in the intestine. Blood, either clotted or fluid, may also be passed in a pure state. Its source is usually the lower part of the bowel, though, when peristalsis is greatly augmented, it may come from the small intestine, as in typhoid fever. Pus may occasionally be recognized macroscopically. "For the detection of small concretions the stools should be passed through a sieve; large concretions are easily recognizable. I generally add some ether previously, in order to somewhat overcome the bad odor" (Ewald).

(b) *Microscopic Examination.*—Diarrheal stools can be examined as discharged, but to solid and mushy dejections a solution of common salt ( $\frac{1}{2}$  per cent.) should be added and all hard masses thoroughly broken up. Different portions of the stools are to be selected for microscopic examination. By the use of the microscope we are enabled to detect parasites and their eggs in the intestinal contents; also mucus in the form of shining, vitreous, homogeneous, or whitish masses; and in the interior of the latter bacteria, various crystals, and intestinal epithelium may be seen. Remnants of vegetable food may simulate mucous islets, but the former strike a blue color on the application of the potassium-iodid test. Microscopically, diarrheal stools show undigested muscle-fibers, fat-crystals, vegetable cells, starchy granules, and innumerable bacteria. On microscopic examination of the dejections in constipation we find "a copious detritus of brown or black color, usually numerous colorless or slightly tinged triple phosphates (phosphate of ammonium and magnesium crystallizing in the form of a coffin-lid), or, more sparse, crystals of neutral phosphate of lime. Seldom do we meet with the rhomboid plates of cholesterin, which are recognized in that they are colored from a reddish-brown to violet by dilute sulphuric acid (1:5), and become blue or green on the further addition of a solution of iodine. Needle-shaped crystals of fat, single and also in the forms of tufts, are frequently met with. Bile-pigment cannot be detected. Undigested remnants of food are only sparsely present, owing to the long detention of the fecal matter in the bowel. Epithelium from the mucous membrane, pus-cells, and blood-corpuscles, unless they come from the passage of the fecal mass through the anus (in which case they are simply adherent to the external surface of the scybala and are but little changed), are greatly altered; they are fatty, degenerated, shrunken, and hardly recognizable. "The micro-organisms are numerous, in lively motion, but have no specific significance" (Ewald).

(c) *Chemical Examination.*—The presence of bile-pigment is easily detected by the Gmelin reaction. The stools must, if needful, be ren-



dered fluid by the addition of water, then filtered, and the filtrate allowed to dry. At the margin of the drop the characteristic green color will appear. Urobilin strikes a red color. The stools in diarrhea may contain ferments capable of digesting albuminoids. The fatty acids are distinguished from fatty soaps by the solubility of the former in ether.

To detect a very small amount of blood that may be intimately mixed with the feces the test of Fr. Müller and Weber gives the most reliable results. Solid or mushy stools are first rubbed up with water and filtered. A portion of the filtrate of liquid feces is added to 5 cubic centimeters of glacial acetic acid and ether, and the whole well shaken. The ether generally settles clear, but if it does not a few drops of absolute alcohol are to be added. The presence of blood (hematin acetate) is shown by a reddish-brown tint given to the layer of ether.

(d) *A bacterial examination* of the intestinal contents, and particularly of any mucus or mucus that may be discharged, may decide the diagnosis of certain intestinal disorders (tuberculosis, amebic dysentery). For the method of carrying on these investigations the reader is referred to special works on diagnosis and bacteriology.

**Physical or External Examination.**—*Inspection.*—A few of the points of greatest diagnostic importance only can be given here. Localized prominences are to be noted over the abdomen, though the fact should be remembered that these may be simulated by localized contractions of the various abdominal muscles. The influence of respiration on these circumscribed bulgings is also to be observed. In the absence of unusual tension of the abdominal walls it is of great value to inflate the large intestine with air *per rectum*, and to note the progressive distention of the intestinal coils as a means of detecting obstructing lesions in the bowel; the position and mobility of a tumor should also be noted. It is often of marked aid to inspect the mucosa of the large intestine, as far as may be, by the use of approved specula (Sims, Ferguson, Cusco, as modified by Ricord).

*Palpation.*—This is the most important method employed in the diagnosis. The patient should occupy the dorsal decubitus, with the head raised, the thighs drawn up, and the mouth open, so as to relax the abdominal muscles. Something may be gained in this direction by distracting the patient's attention. I have found that placing the patient in the lateral decubitus, with the thighs flexed on the abdomen, has enabled me to determine better than in any other manner the degree of mobility of certain tumors. The examiner should not fail to remember the knee-elbow position in cases in which it is desired to palpate the parts occupying the bottom of the pelvic cavity and all deep-seated, movable growths. In certain cases relaxation of the abdominal muscles is only obtainable by bringing the patient under the influence of an anesthetic, and I do not hesitate to do this in cases in which a correct diagnosis is highly important. In palpating the abdomen for abnormal conditions we must keep in mind steadily the relations of the different parts of the intestines, and also the fact that the latter may vary considerably in position—a fact particularly true of the transverse colon (*vide* Enteroptosis). In this connection Ewald's statement "that abnormally situated organs or neoplasms of parts other than the intestines

will, under the pressure of the intestines filled with air or water, return to the position that the organ normally occupies," should be emphasized. He continues: "Tumors of the kidneys or spleen will be pressed up under the diaphragm, tumors of the liver and of the stomach will be forced upward, and those of the large omentum toward the front and downward, while retroperitoneal tumors of the pancreas, of the spinal column, or of the pelvis will remain fixed."

The palpation of pathologic conditions of the intestines will be considered in connection with the separate intestinal affections.

*Percussion* detects a fluid effusion either in the general peritoneal cavity, the position varying with the position of the body, or in circumscribed localities; the latter must not be confounded with areas of dulness that are occasioned by splenic and hepatic enlargements, solid new-growths, or abscesses. Air in the peritoneal cavity (*meteorismus peritonei*) generally gives a pure tympanitic note, though if the tension be very strong, a non-tympanitic tone is elicited. These sounds are general, even extending up to the fifth or fourth rib, and hence they cover the regions of the spleen and liver. The best results when the abdomen is not tense, however, are obtained after inflation of the large intestine with air, since the presence of new-growths, of dislocated viscera, and the relation of the large to the small intestine can thus be accurately determined.

*Auscultation*.—Noises are often audible either at a distance or by means of a stethoscope applied to the abdomen. They are sometimes occasioned by the natural peristaltic movements or by certain voluntary or involuntary spasms of the abdominal muscle. Again, a large amount of air and gas in the intestine may excite sounds. These are heard most frequently in the ileo-cecal region. I have repeatedly confirmed the observation of Ewald, who frequently found in those suffering with chronic intestinal indigestion a swashing or splashing noise, sounding as though air and water were being forced through a narrow space in the ileo-cecal region. These sounds may rarely be found in healthy persons. Similar noises sometimes have their seat in the descending colon, particularly if the bowel is unnaturally dilated by air or fluid. They are often audible prior to an evacuation in cases of colitis. Noises may also originate in the transverse colon, and to discriminate these it is necessary to empty the stomach if we would avoid confusion with identical gastric sounds. Direct auscultation of the intestines renders audible the peristaltic movements, and the absence of the latter indicates paralysis of the intestine, which may be local or general. Friction-sounds tend to appear, and are audible when inflammatory exudates are present. When obstruction of the large intestine is suspected, auscultation should be practised while air is being forced into the rectum, inasmuch as the degree of permeability can be thus determined. Metallic tinkling and amphoric noises may be audible, particularly on making auscultatory percussion, but these are without real diagnostic value.

## ENTEROPTOSIS.

**Definition.**—The descent of the intestines from their normal position. The condition occurs coincidently with gastropstosis, neph-

roptosis, and prolapse of other viscera, constituting splanchnoptosis (Glénard's disease).

**Etiology.**—It is linked with gastropptosis and other forms of ptosis by common etiologic influences, such as sex (being most common in females), tight-lacing, traumatism, muscular strain, numerous pregnancies, rapid emaciation, and probably the wrong use of cathartics. Either the small intestine alone or the large, or both, may be involved. Prolapse of the colon (coloptosis) is the more common, and, according to C. Meinert, is even more frequent than gastropptosis. Lying immediately above the symphysis pubis, it is sometimes elongated and tortuous—"S- or M-shaped."

**Symptoms.**—The condition, even when pronounced, may exist without symptoms. On the other hand, in the majority of instances the intestinal, gastric, and other bodily functions are disturbed, and yet enteroptosis is usually overlooked. Chief among the intestinal symptoms is *excessive flatulence*; not rarely, also, there is membranous enteritis, the latter probably being due to the flexures that produce an arrest of fecal masses, and this in turn causing inflammation (Boas). *Constipation* generally prevails, and sometimes alternates with diarrhea. The symptoms of gastropptosis and nephropptosis are often associated; they are loss of flesh and nervous symptoms, and the latter may simulate those of neurasthenia or hysteria.

The **diagnosis** is made upon the afore-mentioned points and upon the results of a careful physical examination. The position of the colon may be determined by inflation with air or gas. Again, after the injection of water (f5viss-ixss—200-300 c.cm.) a splashing sound is audible; this is double the amount of water required in the normal condition. Glénard has pointed out that a transverse cord (which he believes to be the colon) can be felt in the upper part of the abdomen. Boas and Ziemssen assert that this cord is the pancreas, rendered palpable by the sinking of the stomach.

**Treatment.**—The bowels must be moved regularly, the tonicity of the abdominal walls must be increased by electricity, massage, and hydrotherapy, and in strongly nervous cases the treatment of neurasthenia, including the Weir Mitchell rest-cure, must be instituted. Supporting bandages have been found serviceable. The medicinal treatment aims at meeting certain symptomatic indications, such as flatulence and fermentation.

## INTESTINAL CATARRH.

(*Catarrhal Enteritis; Muco-enteritis.*)

**Definition.**—A catarrhal inflammation of the mucous membrane of the whole or of any anatomic division of the intestinal tract. It may be either acute or chronic, primary or secondary. The chronic variety occurs less frequently than its counterpart, chronic gastritis, particularly in adult life.

**Pathology.**—The morbid lesions of the acute variety do not differ



essentially from those met with in catarrhal inflammation of any other mucous membrane. The first stage is characterized by swelling and dryness of the mucosa: this is soon followed by a copious secretion of mucus, and more rarely of pus, which bathes the membrane more or less completely. After an abundant secretion is poured out the membrane appears rather pale, though the tips of the valvulae conniventes in the small intestines may appear reddened. The solitary and agminated glands, as well as Peyer's patches, stand out prominently, owing to their corrugated condition. Quite often the apices of the solitary glands undergo a necrotic change, thus forming follicular ulcers. The remainder of the mucous membrane may also be the seat of rather extensive areas of superficial erosion, though this must not be confounded with postmortem softening of the epithelium. Postmortem softening of the mucosa, with swelling and even desquamation of the epithelium, is commonly seen at the autopsy. In chronic intestinal catarrh the mucosa presents a slaty hue, with a more or less dark pigmentation of the villi and follicles; it is in most instances thickened, owing to an increase in its connective-tissue elements. In a smaller number of cases it is thinned, particularly in the intestinal catarrh of children, on account of atrophic changes affecting chiefly the glandular and muscular layers. Roughening of the inner surface of the bowel, due to projecting glands, is frequent in those forms of chronic intestinal catarrh that are attended with thickening of the coats. Polypoid cysts may develop in long-standing cases.

**Etiology.**—The **primary** form is produced by (*a*) local irritants, either mechanical or toxemic, that find their way into the intestinal canal. The chief source of these excitants is an unsuitable dietary, and especially is this the case in children. It is readily seen from this fact why the stomach and the intestines are often simultaneously involved in a catarrhal process. (*b*) Over-eating may be productive of the disease, though this often excites diarrhea by merely increasing intestinal peristalsis. (*c*) Idiosyncrasy has a positive influence, the ingestion of certain substances not difficult of digestion being invariably followed by this affection in individuals thus predisposed. (*d*) Toxic substances, whether in the form of tainted food-stuffs (spoiled meats, ice-cream, beer) or inorganic poisons (mineral acids, caustic alkalies, mercury, arsenic) or irritating cathartics, often produce intestinal catarrh. (*e*) Impure water. (*f*) Atmospheric changes, particularly a prolonged high or a sudden fall of temperature, the latter being especially apt to cause it in children. (*g*) An excess or a lack of biliary secretion. Two functions of the bile (its antiseptic properties and its power to stimulate peristalsis) must not be forgotten: the one explains how a paucity of this secretion favors the abnormal processes of fermentation that are capable of exciting catarrh, and the other makes plain the possibility of a bilious diarrhea being due to an excessive hepatic secretion. It is not clear, however, that the latter condition is attended with an actual catarrhal process. The same is true of diarrhea due to fright, excitement, or other nervous influence.

**Secondary or complicating** forms are caused—(*a*) By direct extension from adjacent organs (*e. g.* gastritis, peritonitis, hernia, and invagination); (*b*) By general infectious processes (septicemia, pyemia, typhoid fever, dysentery, cholera, tuberculosis, pneumonia).

The *chronic forms* are met with—(*a*) In certain cachectic states (car-

cinoma, chronic malaria, chronic Bright's disease, Addison's disease, and profound anemia); (b) In connection with disturbances of the circulation, particularly such as produce stasis in the terminal branches of the portal system of vessels: among the chief diseases that tend to prevent the return of venous blood from the intestines are chronic heart-affections, diseases of the liver (especially cirrhosis), and emphysema; (c) Severe cases of chronic diarrhea, supposed to be due to the *protozoon balantidium*, have been reported recently. The evidence to show that the morbid lesions of this disease are of parasitic origin is exceedingly strong. In a case reported by Ortmann, in which the balantidia were readily discovered in the discharged mucus, treatment was of no avail until means were employed that destroyed and caused their disappearance; the patient then made a good recovery.

Among predisposing causes is the *age*, and, though it occurs at all ages, children are particularly liable to the disease. Unfavorable hygienic surroundings, especially when a high temperature prevails, and epidemic and endemic conditions, have a strong predisposing influence also.

**Clinical History.**—From a purely clinical standpoint we recognize not only acute and chronic forms of enteritis, but also a few important varieties based upon their general and local anatomic regions.

The **simple acute form** of general catarrh of the intestines (muco-enteritis) has for its two most characteristic symptoms slight *gripping* or *colicky pains* in the abdomen (sometimes absent), that are followed soon by *diarrheal stools*. The *discharges* consist, at first, of feculent masses, and later of a watery, highly irritating fluid. Diarrhea is due partly to increased peristalsis and partly to the abnormal irritability of the intestinal mucous membrane. Active peristalsis of the intestines may (*vide ante*) be of purely nervous origin, and produce a diarrhea that is to be distinguished from that due to catarrh. The causes that produce the catarrh also produce the undue peristaltic movements—a fact of great clinical importance. If it be true, as physiology teaches, that the stools, owing to the absorption of the watery portions of the food, are normally formed in the large intestines, then catarrh of the small intestines alone cannot excite diarrhea, though it may be attended with increased peristalsis. On the other hand, in *acute colitis diarrhea is conspicuous*, and forms the most important clinical symptom. The vigorous peristalsis also accounts for the gurgling and rumbling sounds (*borborygmi*) that are often felt and heard by the patient himself. These peculiar noises, if pronounced, point to isolated catarrh of the small intestines. The *stools* vary in number from two to ten or more, being increased in frequency after taking food; gases are also formed, causing tympanites. The thin stools either present a bright-yellow or a yellowish-brown color and emit offensive odors. Occasionally they are greenish in color from the presence of considerable quantities of bile-pigment or from bacterial action. In advanced cases of considerable severity there is painful tenesmus; the stools are often small and contain mucus and blood, becoming dysenteric in character, especially when the colon is chiefly affected.

A *microscopic examination* reveals large masses of epithelium and mucus, as well as fungi of many different descriptions, and isolated leukocytes, crystals of calcium phosphate, oxalates, remnants of food (starch-granules, fat, vegetable and muscular fibers). Flakes of yellowish-brown

mucus and large pieces of epithelium may often be seen with the naked eye. The stools give an alkaline reaction, except in cases of acute enteritis in children, when it may be acid (Von Jaksch).

The *physical examination* reveals on *inspection* slight tympanitic distention as a rule. *Palpation* elicits considerable sensitiveness in the majority of cases, though during the colicky pains pressure with the palm of the hand often affords relief. Fluctuation may be detected if the intestines contain much fluid. *Percussion* gives an exaggerated tympanitic resonance, varying, however, with the fulness of the bowel. Nausea may be present, and the appetite is often greatly impaired. There is marked thirst and the tongue is dry and furred.

The *general symptoms* are often entirely wanting, save for a slight feeling of weakness due to the diarrheal discharges. Severe forms of infectious origin often disturb the general health considerably. The patient is languid, and prostration is prominent; he suffers much from headache, and pyrexia is common, the temperature often reaching 100°–103° F. (37.7°–39.4° C.). The higher temperatures are seen among children. Additional evidences of a systemic affection are sometimes observed, such as painful enlargements of certain joints and severe muscular pains.

**Complications.**—The symptoms of gastric catarrh (vomiting, nausea, and pain immediately after feeding) are often associated with those of enteric catarrh; the combination is then spoken of as *gastro-enteritis*.

**Special Forms.**—Though the anatomic limits in the more or less local forms of intestinal catarrh cannot be made out definitely, yet the different clinical pictures observed often enable us to fix the location of the disease with considerable accuracy; it is important, moreover, from the standpoint of the treatment, to accomplish this whenever possible. The following may be briefly described:

(a) *Duodenal catarrh (duodenitis)*, in which form constipation, often obstinate, is present in the place of diarrhea, the colon not being affected; merely local pain, tenderness on palpation, and uneasiness are complained of. These symptoms may frequently be overshadowed by those referable to the stomach when gastric catarrh coexists (*gastro-duodenitis*). Without the presence of jaundice, due to the occlusion of the common bile-duct in consequence of the swelling of the duodenal mucous membrane, the diagnosis of this affection must remain highly doubtful; but, fortunately, this symptom is frequently observed.

(b) *Localized catarrh of the jejunum and ileum* cannot, as yet, be diagnosticated correctly. The condition is often found to be a more or less prominent feature in general enteric catarrh, in which complaint diarrhea is a prominent symptom. The existence of this special variety may be safely inferred when certain enteric symptoms are combined with marked gastric disturbance. Under these circumstances the symptoms indicative of inflammation of the small intestines are rumbling noises (borborygmi), colicky pain, swelling, and slight tenderness over the abdomen in the vicinity of the umbilicus or over other regions occupied by the small intestines. Finally, an *examination of the stools* furnishes valuable points for differential diagnosis. It must be kept in remembrance that in catarrh of the small intestines the stools may be quite



solid, despite the increased peristalsis caused by the catarrhal process (*vide ante*). More frequently, when the ileum is the seat of catarrh the colon is also implicated, this combination being attended with diarrhea, even if it be of minor severity. The thin stools "contain food-remnants, that point indubitably to implication of the small intestine." As the result of increased peristalsis of the small intestines their contents are passed into the large bowel with undue rapidity; hence the latter contains undigested food-constituents and other substances that are normally found in the small intestines. These pass from the rectum unchanged. They are mainly starch, fat, and masses of meat-fiber, the latter of which may be of sufficient size to be seen by the naked eye. This would be pathognomonic evidence of the form of catarrh in question if it were not true that increased peristalsis of the small intestines, due to other conditions, as anemia, extreme nervousness, and fever-conditions, that are not seen in ileo-jejunal catarrh, causes the same fecal peculiarities.

In health the contents of the small intestines give the characteristic color-reaction for bile-pigment, whilst the contents of the large bowel and the stools do not. In intestinal catarrh, with increased peristalsis of the small and large intestines, there is, however, quite often a large admixture of undecomposed bile-pigment (Strümpell) that responds to Gmelin's test,<sup>1</sup> a fact of considerable value in diagnosing catarrh of this portion of the intestinal canal. Nothnagel has called forcible attention to the fact that round bile-stained stools and small pigmented masses of mucus are met with, and are highly characteristic of the diarrhea that marks catarrh of the small intestines.

(c) *Colitis*.—The joint appearance of abdominal pain and diarrhea is almost pathognomonic of this condition. These symptoms, in the absence of the more prominent and above-mentioned clinical features that have special reference to inflammation of the small intestines, point to the fact that the large intestines are the chief seat of the disease.

*Physical examination* is only partially confirmatory of the rational symptoms. The chief sign is tenderness on palpation over the track of the colon. An *ocular examination of the stools* furnishes important practical results. They may contain blood and mucus, and the latter often in masses large enough to be readily visible to the naked eye; it is not intimately mixed with the feces, as in catarrh of the small intestines, but forms separate masses. The feces are often of the consistence of soup. "If the catarrh affects the lower portion of the large intestine chiefly, it may be that the intestinal contents are already formed" in firm lumps, which may sometimes be wholly or partly enclosed in a layer of mucus (Strümpell).

Such *general symptoms* as loss of flesh, weakness, and sallowness of the skin are often observed. Simple diarrhea, lasting but a few days, as a rule, is to be classed with catarrh of the large intestines, since these affections imply increased peristalsis of the large bowel. It is not always easy, however, to discriminate diarrhea due to purely functional influences or to catarrh of the rest of the intestinal tract.

<sup>1</sup> This consists in bringing a few drops of nitric acid in contact with the intestinal contents, when the characteristic play of colors appears. (See also Methods of Diagnosis, pp. 786-789.)

(d) *Proctitis*, or inflammation of the rectum, is characterized by painful tenesmus and by the presence of large quantities of mucus and pus, particularly in the dejections. The disease may be primary, though more often it is secondary to morbid lesions either in organs that are adjacent to or in the rectum itself.

**Chronic intestinal catarrh** may, comparatively rarely, be a *primary disease*, developing gradually. It may also be *secondary* (*vide* Pathology) at times to one or more attacks of acute intestinal catarrh. Generally there are no other local symptoms to call attention to the condition than chronic diarrhea. More rarely there are in addition colicky pain and tenderness over the abdomen. The diarrhea often alternates with constipation, and this is most apt to be the case when the disease is of idiopathic origin and affects only the large intestine (Nothnagel). *Constipation* is constant in those cases in which *atrophic alterations* occur in the glandular and muscular coats, as well as in those in which the lesions are in the small intestines. When constipation is not present the stools are thin, pale, sometimes fermented, emitting offensive odors, and vary greatly in number and quantity. There is commonly present visible mucus, owing to the fact that the most frequent seat of the disease is the large intestine. When the small bowels are also implicated, food-remnants are found in the dejections (*licenteric diarrhea*). That form of diarrhea occurring in *organic diseases of the heart, liver, and lungs* demands brief special mention. Here the serum of the blood is made to exude into the intestines, owing to mechanical obstruction to the return of the venous blood, and this results in a liquefaction of the feces. The stools are apt to be most copious and numerous during the morning hours. Sometimes an irresistible desire to evacuate the bowels seizes the patient as soon as his feet strike the floor on rising in the morning; two or more serous discharges follow each other at short intervals. Subsequently, all discharges cease until the following morning, when the same symptoms are repeated. The general nutrition suffers visibly in chronic enteritis, and emaciation eventually becomes pronounced. I have also noticed slight pyrexia, especially in the evening hours.

**Differential Diagnosis.**—Among the diseases likely to be confounded with acute catarrh of the intestines are *typhoid fever*, *dysentery* (diseases in which diarrhea is a cardinal symptom), *peritonitis*, and *colic*. The chief differential features between simple colic and enteric catarrh may be contrasted thus:

ENTERIC CATARRH.

COLIC.

Diarrhea is constant.  
Fever may be slight or marked.  
Pain is gripping, and is then followed by the stool.  
Tenderness in the intervals between pains.

Constipation is present.  
No fever.  
Pain is colicky, more severe, and is not followed by diarrheal discharges.  
No sensitiveness on palpation.

From *peritonitis* we may readily distinguish catarrh of the intestines by the more intense pain and tenderness, by the constipation, the greater tympany, the constitutional disturbance, and more especially by the anxious face, thoracic respiration, and immobility of the patient, all of which characterize the former disease. When the characteristic

symptoms of *typhoid fever* (the typical temperature-curve, swelling of the spleen, and eruption) and *dysentery* (scanty, frequent stools and tenesmus) are present, they are easily separable from enteric catarrh. In children, however, the diagnosis between typhoid fever and simple catarrh of the bowels offers considerable difficulty; but the temperature-record, if carefully kept, the enlargement of the spleen, and the characteristic eruption, the Widal-reaction, when taken jointly, will warrant the diagnosis of typhoid fever and exclude acute enteritis.

In diagnosing chronic intestinal catarrh we may have difficulty in eliminating *lardaceous disease of the bowels* and *ulceration*; the manner of doing this in case of the latter condition will be pointed out hereafter. *Amyloid degeneration*, however, is a general disease, affecting primarily other organs than the bowel, and hence lardaceous diarrhea is always preceded by the clinical indications of the disease in other parts of the body. The condition also gives a definite etiology as a rule.

**Prognosis.**—The prognosis in uncomplicated cases is favorable, though the possibility of a merging into the chronic form must be borne in mind. Occurring in weakly subjects, especially at the extremes of life, and in the course of debilitating affections, *acute catarrh* of the intestines may become a source of danger to life. Its duration varies much—from three to ten days or more—according as the type of the individual case is mild or severe.

The prognosis in the *chronic forms* is moderately good as to life, though as to cure it is not so, the disease often lasting for many years together, or as long as the chronic conditions producing it remain unremoved. It sometimes exhausts the system of those suffering from serious causal affections of a chronic nature, and occasionally it ultimately proves fatal. The prognosis will depend largely upon the character of the etiologic affection, but intestinal catarrh invariably renders the prospects of life more gloomy.

**Treatment.**—Respecting the treatment of this affection the views of the profession have undergone many changes, even within recent years; hence it may be reasonably inferred that our present therapeutic methods are by no means satisfactory.

**Hygienic and Dietetic Management.**—If the cause be some error of diet, all injurious articles must be rigidly prohibited. In the milder cases due to this cause a mild purgative, followed by proper *dietetic treatment*, is all that is required. Albuminous food in liquid form, such as skimmed milk, weak broths, and even semi-animal articles of diet, as eggs, oysters, sweet milk with seltzer, are usually well borne. In the severe forms predigested liquid foods only should be allowed. When the chief seat of the disease is in the large intestine, we may allow easily digested starches and certain green vegetables (arrow-root, sago, lettuce, water-cress); the coarser vegetables, all fats, and most fruits should be withdrawn absolutely. *Rest* in bed is especially beneficial in that it serves to keep the abdomen warm and mitigates the pain and diarrhea, and, in short, cures the disease. Sinapisms should be applied at the outset until the skin is reddened, succeeded by light linseed poultices until the local sensitiveness has, in a great measure, subsided; after this a flannel band may be applied. The local abstraction of blood by a few leeches, applied to the abdomen or anus, is beneficial in the



early stages in severe types of enteric catarrh, provided the patient's strength is good.

**Medicinal Treatment.**—It is sound practice to prescribe a mild cathartic (castor oil, calomel, or rhubarb, followed by a saline) with a view to getting rid of irritating intestinal contents. Combined gastric lavage and high intestinal irrigation has recently yielded excellent results in my hands; it is an appropriate method of overcoming the fermentative processes that tend to excite and maintain the condition.

If the chief tenderness be localized in the right iliac fossa, corresponding to the course of the colon, a simple enema, slowly given, will stimulate the bowel sufficiently and cleanse it more effectually than a cathartic. Subsequently, chief reliance is to be placed on intestinal antiseptics and astringents, though it must be recollected that the selection of internal remedies must, in part, be influenced by the etiologic indications. For instance, if the cause has been exposure to cold or wet, besides the efforts directed at the local condition diaphoretics and febrifuge mixtures are serviceable. I have found the following combination to be of benefit in controlling the inflammatory action :

R̄. Salol,	ʒss (2.0);
Creasoti,	℥x (0.666);
Bismuthi salicylat.,	ʒj (4.0).
M. et ft. capsulæ No. xx.	
Sig. One every three hours.	

If pain be troublesome, opium or phenacetin may be combined with the above formula, or the following may be employed :

R̄. Argenti nitrat.,	gr. ij (0.129);
Ext. opii,	gr. iss (0.097).
M. et ft. pil. No. xij.	
Sig. One every three or four hours.	

In many instances the secretions of the intestinal tube are decreased for a considerable period after the most active symptoms have been subdued. Here we must supplement the natural juices of the bowel, as follows :

R̄. Pancreatin,	ʒj (4.0);
Sodii bicarb.,	ʒij (8.0).
M. et ft. chart. No. xij.	
Sig. One an hour after meals.	

In cases in which the large intestine is chiefly affected, and when the condition does not yield to internal medicines, treatment per rectum should be employed. If colicky pain be severe, morphin (gr.  $\frac{1}{8}$ —0.008) should be given hypodermically in addition to the measures before suggested. If the diarrhea shows no tendency to abate after forty-eight hours of the general treatment above outlined, large doses of bismuth (gr. xxx to lx—2.0 to 4.0) every three or four hours should be tried. In my own hands lead acetate (gr. ij—0.129), with the extract of opium (gr.  $\frac{1}{8}$ —0.008) in pill-form, has proved a most efficient combination. When there is reason to suspect that the main lesion is in the large bowel, small enemas of starch-water (ʒij—64.0), with laudanum (℥ xx

—xxx—1.33–2.0), every four to six hours, are efficacious. The thirst is best relieved by chipped ice in small quantities or by carbonic acid and Apollinaris waters. For distressing flatulence we may prescribe the alkaline carbonates, or spirits of ammonia, and some carminative.

In chronic catarrh of the intestines the local treatment is of paramount importance. Daily irrigation of the bowel with a weak solution of some antiseptic agent, as salicylic acid (gr. v– $\bar{3}j$ —0.324–32.0), boracic acid (gr. x– $\bar{3}j$ —0.648–32.0), creolin (m v– $\bar{3}j$ —0.324–32.0), or with some such astringent as tannin (gr. v– $\bar{3}j$ —0.324–32.0), or finally with an alterative, such as silver nitrate (gr.  $\frac{1}{4}$ – $\bar{3}j$ —0.016–32.0), will be found to be beneficial. The latter solution is a most excellent remedy, but sometimes excites pain if used in excessive doses. To obviate this, I have often used a mild antiseptic or astringent with the foregoing, giving them on alternate days, and thus obtained most happy results. The only appliance needful is a fountain syringe with a soft-rubber end-piece, which should be gently introduced for a considerable distance into the bowel. The fluid used should be warmed to 90° F. (32.2°C.), and the quantity administered at each sitting should be not less than 2 to 3 pints (1–1.5 liters); this should be allowed to flow in slowly. The patient should, as a rule, assume the dorsal decubitus, though if the fluid is to be carried as high up as possible, the knee-elbow position may be assumed or the patient may be placed on the left side with the hips elevated.

The same careful attention must be paid to hygienic details, and especially to the diet, as is directed in the acute form. In addition, flannel should be worn next the skin both in winter and summer. If the strength will admit of it, cold baths are useful.

A stay at a suitable spa (Saratoga, Bedford, Virginia Springs, Carlsbad, Kissingen) often produces most satisfactory results.

Among internal agents, zinc oxid (gr. v to x—0.324–0.648—t. i. d.), silver nitrate, lead acetate, and alum, given with tonics, such as strychnia, arsenic, and iron, are especially to be recommended.

The management of this troublesome malady depends upon the indications furnished by the causal chronic affections. No method of treatment can succeed, however, that is not carried out patiently, systematically, and over long periods of time.

## DIARRHEAS OF CHILDREN.

### ACUTE GASTRO-INTESTINAL CATARRH.

(*Acute Gastro-enteric Infection; Summer Diarrhea; Gastro-enteritis; Cholera Infantum; Mycotic Diarrhea.*)

**Definition.**—This is the usual intestinal trouble that prevails during the warm summer months. It usually takes the form of an epidemic, and its course is manifested by a sudden onset, high fever, irritability of the stomach, frequent watery evacuations, symptoms of nerve-involvement, and possible collapse in young children. This form of diarrhea

usually follows an attack of acute indigestion, in which it very frequently has its origin. Acute gastro-intestinal catarrh stands midway between acute indigestion and ileo-colitis.

**Etiology.**—Two important conditions seem to be necessary to influence the disease—temperature and diet. Nearly all the fatal cases occur in the artificially fed, and a general and well-recognized belief associates special danger with the second summer of children. Out of nearly 2000 fatal cases collected by Holt, only 3 per cent. were exclusively breast-fed. Generally speaking, the disease has its origin in some irregularities in artificial feeding. Heat is an important element in the continuation of the disorder when once commenced.

The death-curve begins to rise in May, increases during June, climbs to the highest point in July, and very greatly declines during August and September. High temperature must not, however, be regarded as the sole or direct agent, but only one of several factors.

**Bacteriology.**—Ballard believes the cause to be a micro-organism (not yet isolated) that is constantly present in the superficial layers of the earth; it enters the food, and develops under favorable conditions—either inside or outside of the body—a virulent poison, or ptomain, that gives rise to the symptoms seen in the disease. This unknown micro-organism is supposed to play the same part in producing the disease as the comma bacillus in Asiatic cholera.

Baginsky, experimenting with the micro-organisms formed in the stools of infants suffering from diarrhea, failed to find any that could be regarded as specific or pathogenic, but found many saprophytic or non-pathogenic bacteria; he inclines to the belief that the decomposition-products formed by these different varieties of micro-organisms are the toxic substances that give rise to the disease.

Meinert, while believing that micro-organisms and their resulting ptomains may give rise to an intestinal catarrh, believes that the acute forms of summer diarrhea are produced directly by the action of a high temperature—*i. e.* a sort of heat-stroke, having nothing to do with micro-organisms or ptomain-poisoning.

Although no pathogenic organisms have been isolated, this does not disprove their existence; and, on the other hand, we are not yet in a position to accept the conclusion that summer diarrhea is a definite parasitic disease, like Asiatic cholera or scarlet fever. A high atmospheric temperature continuing for days and nights favors the development of all forms of saprophytic organisms that grow in all kinds of food, animal and vegetable, and under favorable conditions produce poisons such as *muscarin*, which when taken into the stomach gives rise to fever, depression, and collapse.

The *proteus* class of bacteria are most frequent, and are most likely to possess pathogenic properties, according to Barker, who has made a complete study of the subject. With him are in accord the opinions of Jeffris and Baginsky.

**Pathology.**—A catarrhal swelling of the mucosa of the large and small bowel is present; the mucosa itself is pink in color from capillary congestion. Peyer's patches are enlarged. The whole intestinal tube shows an early stage of inflammation (ileo-colitis). In addition there is most likely some involvement of the sympathetic nerves, leading to



dilatation of the capillaries and transudation of serum into the intestine, and to alterations of the pulse, temperature, and respiration. Its nature is paralytic, and closely resembles in its results experimental sections of the sympathetic nerves. The changes in the other organs are slight. Broncho-pneumonia frequently occurs. The spleen is often swollen, the brain is anemic, and the kidneys are congested.

**Symptoms.**—Clinically, we recognize two forms of acute enteric infection: (1) acute dyspeptic diarrhea, and (2) cholera infantum.

(1) *Simple Gastro-intestinal Catarrh (Acute Dyspeptic Diarrhea).*—The child may appear in its normal condition, with merely an increase in the number of stools, with or without fever; restlessness is usual at night. This condition may continue for two or three days, when the stools become more frequent and offensive, containing undigested food and curds. The *odor* by this time is very pronounced—penetrating and adhering to the clothes and room for a long time. Frequently the disease has a sudden onset, with vomiting, griping pains, and fever which may quickly rise to 104°, 105°, or 106° F. (40°–41° C.). Convulsions may be the commencement of the attack. The abdomen is sensitive and swollen, and the child lies with its legs flexed on the stomach. The *stools* consist of grayish or greenish-yellow feces (mixed with gas, curds, portions of undigested food), and some fluid. In children two years of age and older the stools may contain unripe fruit or very large curds from excessive drinking of milk. Relapses are frequent, and during hot weather the frequency of the attacks may lead to the commencement of a severe entero-colitis.

In delicate children a severe attack, especially if it is accompanied by convulsions, may prove fatal. From the fact that the general symptoms may be few, the case is often allowed to go on for several days, under the impression that the child is “only teething.”

(2) *Cholera Infantum.*—The initial symptoms are sudden. The child voids immense stools, at first fecal, if no preceding diarrhea have been present. Soon they become watery, light yellow or greenish in color; frequently they are so thin and colorless as to pass through the napkin without leaving a stain. At times they contain a few yellow or greenish flocculi or a mass of mucus, and in all cases they are odorless. Very often the stools are brown and liquid, with a small quantity of fecal matter, having a peculiar musty odor that clings to the napkin and child for days. The number of stools per diem may vary from six to thirty, and a most remarkable feature is the fact that they are evacuated with considerable force.

The *stomach* becomes irritable, refusing everything; even ice is rejected as soon as swallowed. The *appetite* is, of course, entirely lost; intense thirst prevails, the little patient drinking at every chance and following the receding glass with eager eyes. The *tongue*, moist at first, soon becomes dry and pasty; the abdomen is collapsed. The *temperature* is always high—105° or even 108° F. (40.5°–42.2° C.); and the *pulse* small and very frequent—130 to 180 beats per minute. The *breathing* is shallow and irregular, and the *eyes* anxious and staring, but soon becoming dull. The urine diminishes in quantity daily.

With this array of symptoms there is a striking and appalling change in the child's general appearance. Within a few hours smiling, perhaps

plump and rosy, it can now scarcely be recognized; the face has become pale and pinched, the eyes and cheeks sunken, the eyelids and lips wide apart from loss of muscular control, the muscles flabby, the bones prominent, and the skin greenish or cadaverous, hanging in loose folds from the wasted frame, all the fat having melted from the body.

*Collapse* comes on soon: the hands, feet, nose, and breath become cool, the respirations more unequal, and there are drowsiness and utter apathy. When life is near its close, vomiting stops, the whole surface becoming cool and clammy as the patient sinks into a state of coma, with injected eyes and contracted pupils. At last the end is reached quickly, preceded perhaps by a slight convulsion. The *duration* of the disease is short; it may prove fatal in from one to four days.

**Diagnosis.**—This is readily made. There is no other intestinal trouble in children to mislead one. The character of the stools, the extreme irritability of the stomach, the disturbed respiratory rhythm, high temperature, intense thirst, constant vomiting, frequent watery stools, and collapse soon coming on, tell the true story.

The **prognosis** is very unfavorable.

**Treatment.**—The treatment of *acute gastro-intestinal catarrh* divides itself into hygienic, dietetic, and medicinal measures. If a child is attacked in the city during the summer and does not yield to treatment in two or three days, it should be sent to the country or seashore. In the case of a child under two years this is absolutely imperative. Fresh air is important in all diarrheal disorders in summer both in country and city, and all cases should be kept out of doors as much of the time as possible. Children should be kept quiet—not permitted to walk, even if able. Bathing is soothing and beneficial in that it ensures cleanliness and, what is very important, reduces the temperature.

*Dietetic treatment* is of great importance. It should be remembered that digestion is arrested in the early stage, hence all food must be withheld; to give food at this stage is to do harm. Thirst may be controlled by ice- or albumin-water, toast-water, or gum-water, with a little brandy.

*Medicinal Treatment.*—The first step is directed against the acute indigestion and the active putrefaction going on in the intestinal tube. The indication, therefore, is to empty thoroughly the whole alimentary tract as soon as possible, and no other treatment must be thought of until this end has been accomplished. Whenever vomiting persists the stomach should be washed out; usually one washing is sufficient. In older children emetics will favor complete emptying of the stomach, but are never to be given to infants under two years. For the intestine calomel and soda may be used; for the colon we may use, in addition, irrigation: this is advisable in all cases, as it hastens the effect of the cathartic and removes at once much irritating and offensive material. Opium should not be used until the whole intestinal tube is clean, and then cautiously. Spirits of chloroform, or camphor, is a better remedy for the pain than opium in any form. In older children the hypodermic injection of morphin and atropin in appropriate doses most frequently controls the whole train of symptoms. Bowles has used lactic acid in the maximum dose of  $1\frac{1}{4}$  grains every hour in 60 cases of summer diarrhea, and found it to control the symptoms in every case in from 24 to 48 hours.

*Treatment of Cholera Infantum.*—In this form of infection of the intestinal tract we are likely to forget that we are called upon to treat

a case of acute poisoning. The toxic material acts both powerfully and quickly as a cardiac and systemic depressant. It also acts toxically upon the nerve-centers, and paralyzes the vaso-motor nerves. According to Holt, the leading indications are—(a) to empty the stomach and intestines; (b) to supply the body with fluid to offset the great loss by vomiting and purging; (c) to counteract the effect of the poison on the heart and the nervous system; (d) to reduce temperature; and (e) to treat the symptoms as they arise. In the first condition thorough stomach and intestinal cleansing is absolutely necessary. Moreover, we cannot depend on emetics or purgatives to arrest pain and to limit the effect of the poison on the nervous system; a hypodermic injection of atropin and morphin is essential. Morphin must be given with discrimination to young children, especially when the vomiting and purging are slight; it is especially contraindicated when stupor or collapse seems near. Small doses repeated are better than larger single doses. Holt gives gr.  $\frac{1}{100}$  (0.0006) of morphin, with gr.  $\frac{1}{800}$  (0.0008) of atropin, as the first dose in a child one year old. In supplying fluid to the exhausted tissues it is useless to attempt to give them by the mouth, or even by the rectum, as by both avenues it would be rejected. An injection into the cellular tissues of the buttocks, back, or thighs of a saline solution (40 grains—2.59—of common salt to a pint of sterilized water) is the best way to meet the drain. One pint (half liter) may be used every twenty-four hours, and larger quantities may often be used with advantage. Baths must be given to control temperature, and ice-bags should be placed to the head. Ice-water injections will aid in the control of temperature, and ice-suppositories act efficiently when the water is not retained. Stimulants may be given hypodermically. During the active stage nothing should be allowed by the mouth except iced brandy or champagne.

#### CELIAC DISEASE.

(*Diarrhœa Alba; Diarrhœa Chylosa.*)

**Definition.**—A form of intestinal catarrh marked by copious fetid and frothy discharges resembling gruel.

**Pathology.**—Although ulcers have been noted in the intestine, the pathology of the disease is not known. Says Osler: This affection resembles somewhat the disease in adults known as “hill diarrhea” or the “white flux” of India, with which psilosis or sprue, another tropical disorder, is considered identical by some writers.

**Etiology.**—The disease is limited chiefly to children from one to five years old, though it has no connection with an inherited tendency. The *filaria sanguinis hominis* has been found in the feces in cases of diarrhœa chylosa.

**Symptoms.**—The disease is of slow development, and the characteristic feature consists of copious diarrheal (though not watery) stools, resembling gruel or oatmeal-porridge. These are also frothy (*frog-spawn*), and horribly fetid. The physical signs consist of a moderate distention of the abdomen and a boggy sensation that is imparted to the palpating finger. The general features may be summated in gradually increasing emaciation, debility, and pallor.

The **course** is prolonged, and terminates fatally as a rule.



The **treatment** is purely symptomatic, unless the presence of parasites be suspected, when large antiseptic enemata should be slowly administered in a methodic manner at intervals of a day.

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### PHLEGMONOUS ENTERITIS.

THIS is a suppurative inflammation of the submucous layer of the intestines. It is among the rarest of grave maladies, especially as an irrelative disease. It may be diffuse or take the form of a circumscribed abscess. Rarely it occurs as a complicating condition in septicopyemia and in malignant types of the exanthemata, resulting in the formation of abscesses that have their seat usually in the duodenum. Phlegmonous enteritis may be secondary to strangulated hernia or intussusception. The stomach may be similarly affected at the same time.

**Symptoms.**—The local signs simulate closely those of peritonitis, and the position assumed by the patient is identical with that seen in the latter disease. Among the symptoms vomiting is prominent, though not diagnostic; it is always severe, and may become stercoraceous. Pain and tenesmus, when due to obstruction, are intense. Rigors more or less severe have been observed. The temperature is high, and its curve is somewhat typical of the fever of suppuration. The disease is very fatal, and when it is about to terminate unfavorably the patient passes from a condition of extreme prostration to one of utter collapse.

**Treatment.**—The physician's task is confined to an attempt to support the powers of the patient and to relieve his inordinate suffering. The surgeon's aid should be invoked early in cases of obstruction.

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### CROUPOUS OR DIPHTHERITIC ENTERITIS.

**Definition.**—An intense inflammation of the intestinal mucosa, accompanied by a croupous exudate; it occurs in connection with widely various conditions and diseases. It has been definitely shown that if from any cause the epithelial covering is destroyed, the same agents that are productive of croup may set up inflammation in the part.

**Pathology.**—There are two sets of morbid lesions to be distinguished: (1) The first and most important class exhibits a croupous deposit varying greatly in thickness and in superficial area. Its color is variable, being sometimes of a *grayish* or *grayish-white* hue, though more frequently perhaps *grayish-yellow*. I have almost invariably seen these lesions in the colon, while other observers have seen them in the cecum and small intestines. (2) In the second group the solitary follicles alone are inflamed, and the diphtheritic deposit is merely coextensive with their mouths.

The *etiologic* factors may be (*a*) mechanical irritants (impacted feces, enteroliths, gall-stones); (*b*) chemical irritants (ammonia, acids, mercury, arsenic); (*c*) the condition may be secondary to acute infectious diseases and certain chronic complaints (Bright's disease, pyemia, carcinoma).

**Symptoms.**—When mechanical irritants give rise to symptoms, they do not differ from those due to stercoral ulcers, and there is no way of recognizing the croupous deposits unless they be discharged *per rectum* and are detected in the stools. In cases that arise from the action of irritant poisons vomiting and purging are well marked and the dejections contain blood-stained mucus. We cannot be certain about the presence of croupous deposits in toxic cases unless they be found in the discharges. When phlegmonous enteritis occurs as a complicating condition in infectious diseases, the symptoms are almost completely veiled. The symptomatology of the follicular variety cannot be separated clinically from that of follicular ulceration.

The **treatment** is that of the indications presented by the causal conditions or affections in the course of which it occurs.

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## CHOLERA MORBUS.

(*Cholera Nostras.*)

**Definition.**—A self-limiting disease, characterized by a brief course and by serous vomiting and purging, colicky pains, and often muscular cramps.

**Pathology.**—No constant anatomic changes have been noted. So far as observed, they are analogous to those seen in acute gastro-enteritis, though cases have terminated fatally in which no morbid lesions were detectable at the *postmortem* examination.

**Etiology.**—Among predisposing causes, the *age* and the *season* exert the most prominent influence. The condition may appear in subjects under two years, when the term “cholera infantum” is employed,<sup>1</sup> though it is more often met with in older children and adults. It is almost invariably seen during the heated term, from the latter part of June to September, being rarely met with at other seasons, and it is especially prevalent during the month of August. Bad hygienic environments, foul air in particular, have a noticeable effect, and, though not as yet absolutely proved, it may be safely inferred from the clinical history and the usual course of the affection that it is of microbic origin. Among other factors are improper food, particularly unripe fruit, cucumbers, egg-plant, and exposure to cold and wet. Various organisms (especially the Finkler and Prior spirillum) have been found present. No one variety, however, has been definitely found to be the cause of the condition. Virulent specimens of the bacillus coli communis, and even of the streptococcus, have been noted.

**Clinical History.**—The symptoms are those of an intense gastro-enteric catarrh. The *onset* is often sudden, and is marked by abdominal pain, vomiting, and diarrhea. At first the *vomit* consists of food, and later of a mixture of bile and mucus. The *dejections* are fecal in character at the onset; though they soon become watery, and may resemble the rice-water stools of Asiatic cholera.

<sup>1</sup> This affection is described separately (*vide* p. 800).

*Physical examination* reveals only tenderness on pressure over the abdomen, particularly the epigastric region.

*General symptoms* are not wanting. Cramps in the calves are common. The thermometer may register a high temperature, though it varies greatly, ranging from  $100^{\circ}$  to  $106^{\circ}$  F. ( $37.7^{\circ}$  to  $41.1^{\circ}$  C.). The skin-surface, however, and more particularly that of the extremities, feels cool, and owing to this fact the rectal temperature should be recorded. The pulse, as the case progresses, becomes rapid and feeble. The face is pale or even cyanotic, the features looking pinched. The extremities lose their plumpness, and the patient usually appears prostrated and mentally dull. The urine is apt to be scant, high-colored, and sometimes albuminous, and thirst is extreme. Often the picture of general collapse is soon developed.

**Differential Diagnosis.**—The symptoms of cholera morbus resemble so closely those of *Asiatic cholera* as to preclude the possibility of a differential diagnosis. The dissimilarity between these affections lies partly in the fact that no connection can be established between isolated cases of cholera morbus and cases of true Asiatic cholera when the latter disease is not epidemic. A bacteriologic examination of the stools alone, however, permits a certain discrimination. During a cholera epidemic the distinction between them is made without difficulty (see Diagnosis of Asiatic Cholera).

**Prognosis and Duration.**—The duration of the disease varies from three or four hours to two days. It is rarely fatal, though in persons suffering from such chronic affections as Bright's or cardiac disease, and also in the aged, the prognosis is only guardedly favorable. It is said to be more unfavorable when cholera and dysentery prevail (Loomis). A pronounced algid state should not be looked upon as free from danger. I remember two cases attended with profound collapse that recovered, but in which a condition of marked neurasthenia, indigestion, and functional heart-disturbance formed a series of sequelæ that lasted for several months. Nearly all cases, however, recover without sequelæ.

**Treatment.**—The diet must be rigorously restricted, and predigested milk and animal broths are to be prepared as lightly as possible until convalescence has been fairly entered upon. The comfort of the patient, as well as the cure of the disease, is much enhanced by keeping the patient at absolute rest. Local measures are useful in combating pain and vomiting. A large mustard-paste applied to the stomach and abdomen, followed by linseed-poultices that are to be worn constantly, has a strong influence in accomplishing the relief of the symptoms before mentioned. If indigestible substances have been taken prior to the attack, prompt though mild laxatives are to be given at the beginning of the treatment. For the excessive thirst chipped ice, over which a little brandy has been sprinkled, is effective. For controlling the pain, the nausea, and the diarrhea in this disease we have a remedy *par excellence* in the hypodermic administration of morphin. The dose should vary (gr.  $\frac{1}{4}$  to  $\frac{1}{2}$ —0.016 to 0.032) according to the severity of the symptoms, and I have rarely found it necessary to give a second dose. Not only are the pain and diarrhea subdued, but the peripheral circulation is also re-established. It has also been recommended to administer opium by the mouth for these symptoms in the form of the solid extract or laudanum, but the results are infinitely more brilliant



when the drug is employed subcutaneously. The other points in the treatment of this affection are identical with those discussed under the treatment of Gastric and Enteric Catarrh.

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## INTESTINAL INFARCTION.

A FEW instances of occlusion of the superior mesenteric artery by an embolus have been recorded recently. The condition produces hemorrhagic infarction of the small intestines, and is marked by grave and usually fatal symptoms. Its causes are sometimes obscure. The cases that have come to autopsy have shown intense congestion, with a swollen, blood-infiltrated state of the jejunum and ileum. Osler has seen three instances: in one there were numerous vegetations on the mitral valves from which the embolus was probably derived; in another the superior mesentery was plugged at its orifice; and in the third the artery was blocked by a portion of the fibrous clot of an aneurysm of the aorta near the diaphragm. The *symptoms* are urgent. Quite often diarrhea is present from the first, the dejections sometimes becoming blood-tinged. Soon, however the characteristically grave symptoms of intestinal obstruction supervene—viz. great pain, vomiting, and constipation, with excessive tympanitic distention of the abdomen. The condition cannot be recognized from the symptoms and physical signs on account of their close resemblance to the various forms of obstruction, yet its probable existence may be inferred in the presence of one of the known causal conditions.

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## INTESTINAL ULCERS.

### DUODENAL ULCER.

**Definition.**—A small, round perforating ulcer of the duodenum, and a counterpart of the gastric ulcer (*vide* p. 763).

**Pathology.**—The morbid characteristics are so nearly identical in appearance and nature with those of peptic ulcer of the stomach that they scarcely demand a separate presentation. The *seat* of the ulcer is with few exceptions above the orifice of the common bile-duct. When these ulcers heal the resulting cicatrix produces stenosis, which in turn leads to dilatation of that portion of the duodenum back of it, and finally of the stomach also. Progressive cicatricial contraction may completely close the *ductus communis*, and in like manner the pancreatic duct or the portal vein may be occluded. Protective adhesive inflammation between the duodenum and the adjacent parts (pancreas, gall-bladder, liver) often prevents complete perforation of the duodenal wall: when perforation does occur, however, the peritoneal cavity may be opened, causing peritonitis, or a fistulous communication may be established with the gall-bladder, liver, or pancreas. Rarely the direction of

an abscess resulting from perforation is outward, pointing at the seventh intercostal space. In cases in which the posterior wall of the duodenum has been perforated the abscesses burrow through the mediastinum into the tissues of the neck and open posteriorly near the shoulder-blade (Loomis). The concurrence of a gastric and a duodenal ulcer is not infrequent.

**Etiology.**—Though the duodenal ulcer has, as a rule, the same mode of origin as the gastric ulcer, the fact should be prominently mentioned here that extensive burns of the skin-surface of the body are quite prone to be followed by a perforating ulcer of the duodenum, while gastric ulcers are seldom caused in this manner. To explain this form of ulceration of the duodenum is difficult. It is quite probable, however, as in other forms of duodenal and gastric ulcers, that the circulation is arrested by an embolus (from decomposing masses of blood) at some point in the mucous membrane, the acid gastric juices subsequently digesting the part that is thus deprived of its blood-supply. In confirmation of this view we may mention the facts that these ulcers are rarely situated below the point of entrance of the *ductus communis* into the duodenum, and that the contents of the portion of the duodenum above the mouth of the common bile-duct have their parallel in those of the stomach, the acid secretion remaining unchanged until it becomes mixed with the biliary and pancreatic secretions. Sir William Gull suggests that the situation of the ulcer depends somewhat upon the fact that the portion chiefly implicated is so much more fixed than the rest of the organ that one can imagine its surface becoming abraded during peristaltic movements.

The influence of *sex* and *age* as causal factors is notable and in striking contrast with their import in gastric ulcer. In the latter disease most instances occur among young females, while in duodenal ulceration they occur, as a rule, in males between the thirtieth and fortieth years. Of 64 cases collected by Kraus, only 6 sufferers were females. In view of the fact that the pathology of gastric and duodenal ulcers is the same, these differences respecting their etiology are inexplicable. The ratio of cases of gastric and duodenal ulcers, however, is about as 30 to 1 in favor of the former.

**Clinical History.**—Perhaps no real distinction between the symptoms of gastric ulcer and those of its analogue affecting the duodenum can be said to exist in most instances. A probable diagnosis of ulceration of the duodenum has, however, been repeatedly made, and sometimes verified by the subsequent autopsy. Inasmuch as the essential symptom in ulceration of the duodenum—viz. *melena*—occurs not infrequently without the presence of marked gastric symptoms, there is great danger that the disease under consideration will be mistaken for other affections of the intestine in which this is also a prominent symptom. Under like circumstances, if duodenal ulcer be classed with gastric ulcer, there is great danger that the true nature of many cases will be overlooked. The difference in the symptomatology in the two forms of ulceration is owing solely to the difference in locality, implying a difference in nervous and blood supply.

The distinctive features of this disease may be shown by presenting its leading symptoms by the side of those characteristic of gastric ulcer:

## DUODENAL ULCER.

Usually occurs between 30 and 40 years, except when due to external burns.

Males are more frequent sufferers than females, in the proportion of 10 to 1.

Onset marked by intestinal hemorrhage, which may recur at intervals of varying duration.

The melena may be preceded by or accompanied by hematemesis, though not generally.

Blood in the discharges often is bright red, profuse, sometimes dark, and tarry from the action of acid chyme when slight, though less marked than when from the stomach.

Pain may come on late, two to four hours after meals; more often it is absent. It is localized in the right hypochondriac region.

Gastric crises of great violence occur without reference to time of taking food.

Hemorrhage from the bowels is apt to occur at time of crises. Vomiting less frequent.

Jaundice occasionally present from occlusion of bile-duct.

Less marked improvement after diet has been regulated.

Painful point is either in the same areas on the right side or is absent altogether.

## GASTRIC ULCER.

May occur at any age after childhood.

Females are the chief sufferers.

Gastric hemorrhage occurs, preceded by other gastric symptoms, as a rule.

Blood may appear in the stools, usually after hematemesis.

The blood in the dejections is dark and tarry from the action of the gastric juices.

Pain paroxysmal, greatly influenced by taking food; often relieved by vomiting. Pain sharply localized in the epigastric region, about two inches below the ensiform cartilage.

Gastric crises come on soon after taking food.

Vomiting and hematemesis apt to occur at culmination of crises.

Jaundice absent.

Usually a marked improvement follows regulation of diet.

Boas claims to have discovered a painful point over the tenth and twelfth vertebræ, on the left side.

Of the symptoms mentioned under Duodenal Ulcer, the *intestinal bleedings* and *violent crises* (in which the pain is referred to the right hypochondrium, and comes on from two to four hours after meals) are the most diagnostic. While hemorrhage is the leading single symptom in this complaint, we must not, in attempting to estimate its significance in any case, neglect to eliminate hemorrhoids, carcinoma, tuberculosis, dysentery, and finally the hemorrhagic diathesis,—all conditions in which melena occurs as a cardinal symptom. Recently many cases have been reported in which there was an entire absence of symptoms until perforation occurred, followed by rapidly fatal suppurative peritonitis. In regard to these accidents we may refer to what is said in the description of the latter disease (*infra*). The signs of dilatation of the stomach, for reasons before stated, sometimes follow the healing of these ulcers, associated usually with chronic gastro-duodenal catarrh, the latter being due to mechanical causes. Rarely, stenosis of the *ductus communis* takes place as the result of duodenal ulcer; more frequently tumors either compress or occlude the lumen of the bowel below the mouth of the duct. The symptoms presented differ widely from those due to stenosis above the duct, the most characteristic being the continual backward flow of bile into the stomach, sometimes attended by constant vomiting of biliary secretions. As in the case of gastric ulcer, in the duodenal form there is at times so much thickening about the base of



the ulcer as to give rise to the signs of tumor. This is especially true of those instances in which the base of the ulcer becomes attached to adjacent organs; in such cases the resemblance to malignant disease may be striking.

**Prognosis.**—The risk to life is greater than in gastric ulcer, since there is less tendency to cicatrization.

**Treatment.**—The suggestions made in the treatment of gastric ulcer are entirely applicable to the duodenal form also.

**Follicular ulcers** have already been described under Catarrhal Enteritis (*vide* p. 793), and they have a similar pathology and etiology. When present in goodly numbers they give rise to a symptom peculiarly their own, and hence may be dignified by a separate though brief mention. The symptoms of the condition arising in the course of chronic enteritis often escape observation for a long time. The most characteristic manifestation is the appearance in the stools of conical-shaped masses of mucus resembling “boiled sago.” Marked weakness and emaciation rapidly ensue. Among children the disease is common and assumes an aggravated form, the little sufferers quite frequently reaching their end as the result of inanition. An unfavorable termination may be due to perforation followed by suppurative peritonitis. The *treatment* coincides with that of chronic enteritis.

**Stercoral ulcers** are the result of the mechanical effect of hard fecal scybala (often enteroliths, due to a deposit of lime-salts) upon the intestinal mucous membrane. They occupy the sides or tops of the normal folds in the colon.

**Symptoms.**—There is, as a rule, a clear history of *chronic constipation*, though the physician may, notwithstanding, be called on account of the presence of *diarrhea*; this is caused by the retained hardened feces working their way into the rectum. A *digital exploration* will now clear up the diagnosis. There are *tenesmus* and *colicky pain* in the abdomen, the latter symptom being also complained of when no diarrhea is present. The pain often occurs in severe paroxysms that may be attended with the discharge of thready or flaky mucus, pus, and sometimes blood.

**Physical Examination.**—Palpation may in rare instances reveal the presence of a sausage-shaped tumor and sharply localized tenderness over the seats of ulcers.

Enteroliths may lie in the intestines for years together, or they may finally be discharged with the stools. The ulceration that is thus caused often passes unrecognized.

The **prognosis** is good if the condition be not overlooked.

The **treatment** consists in thoroughly evacuating the bowels by salines and simple enemata, persistently used. Subsequently these cases are to be managed in the same manner as other non-specific ulcers of the bowels.

**Simple ulcerative colitis** is a not uncommon complaint, and one that is frequently associated with chronic intestinal catarrh. The ulcers may be quite extensive, removing the greater portion of the mucous

membrane, though in several instances I have observed cases at the Episcopal Hospital that were superficial; these were confined almost solely to the mucosa. The muscular layer of the gut was greatly hypertrophied and its lumen increased in every instance. The non-ulcerated portions of the mucosa looked, in part, quite pale, and in part quite dark. Polypoid growths have been observed situated between the ulcers.

The **etiology** is obscure. The disease is met with most frequently in persons past middle life, and it is quite probable that chronic enteritis sustains a causal relation. Those whose constitutions have been enfeebled by previous disease or an unfortunate hygienic environment are the chief sufferers.

**Symptoms.**—The clinical features are ill defined at the onset, and are often erroneously ascribed to indigestion. *Diarrhea* (lienteric in character) is its most prominent symptom, and with it constipation may alternate. Pus and blood are absent with the rarest exceptions. The general health soon suffers greatly, the patient becoming weak and emaciated.

The **course** of the disease shows it to be of the subacute type, tending in most cases to become chronic.

The **diagnosis**, apart from a consideration of the symptoms above mentioned, requires the elimination of *dysentery*—an easy task as a rule.

**Prognosis.**—This is unfavorable during the earlier stages in the aged. The strong innate tendency of the disease to become chronic must be considered.

The **treatment** embraces (*a*) a careful regulation of the diet, consisting in a restriction of the patient to liquids and semi-solids during the acute stage; (*b*) the administration of a gentle laxative, followed by antiseptics and astringents (bismuth gr. xxx—2.0—combined with salol gr. v—0.324—every four hours); (*c*) the more serviceable local measures in the form of enemata, among the best being silver nitrate (gr.  $\frac{1}{4}$  ad  $\frac{3}{4}$ —0.016 to 32.0) or creolin (2 per cent.).

**Solitary Ulcers.**—"Two instances of ulcer of the cecum, both with perforation, have come under my observation, and in one instance a simple ulcer of the colon perforated and led to fatal peritonitis" (Osler).

The diffuse catarrhal ulcer is inseparable from acute enteritis; the cancerous ulcer is alluded to under the latter head.

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## APPENDICITIS.

**Definition.**—A catarrhal, ulcerative, or interstitial inflammation of the appendix vermiformis. It must be confessed that, according to our present views, appendicitis is a surgical rather than a medical affection, particularly from the standpoint of treatment. Knowing from personal experience and observation, however, that general physicians are constantly meeting with cases of appendicitis, its prompt clinical recognition by the latter is not only a matter of interest, but also of great practical importance for two reasons: First, in order that surgical inter-

vention can be instituted at the proper moment; and secondly, because appendicitis is the leading serious disease of the intestinal tract.

The term "appendicitis" includes the affections *typhlitis* (inflammation of the cecum) and *perityphlitis* (a similar involvement of the connective tissue behind the cecum), for the reason that with few exceptions when the symptoms of the latter affections are presented the appendix vermiformis is the part primarily affected. To the physicians and surgeons of America belongs the credit of having first established the truly important rank of appendicitis.<sup>1</sup>

**Anatomic.**—Without any known function the human appendix vermiformis represents the remains of the enormous cecum of inferior animals, especially rodents and herbivora. Clado asserts that the vermiform appendix is kept in position by two folds of peritoneum, a meso-appendix, which is attached to the iliac fossa, and a second fold, perpendicular to the first, which is attached to the posterior portion of the small intestine.<sup>2</sup> A lymphatic gland generally occupies the angle formed by the appendix, cecum, and the small gut; this receives all the lymphatic vessels of the appendix. The size of the latter varies greatly. Ferguson,<sup>3</sup> after measuring 200 appendices, gave as the average length  $4\frac{1}{2}$  inches (11.4 cm.), and as the diameter, that of a No. 9 English sound—about a quarter of an inch (0.62 cm.). Berry's studies, which are partly based upon personal examination of 100 bodies, and partly upon comparison of his own results with those obtained by other investigators, gives the average length in all the observations as 9.2 centimeters (3.6 inches). The caliber is ordinarily of the size of a goose-quill. Very exceptionally, as in a case reported by Swan, there is a congenital absence of the appendix. Its two fibro-muscular coats (external longitudinal and internal circular) are thick; its mucous membrane contains lymphoid elements in abundance. The blood-supply is derived from the ileo-colic artery at the valve, a single branch running to the end of the appendix. Shortly after middle life the cavity of the appendix becomes obliterated. Its blind extremity points most frequently toward the spleen. The appendix may lie behind the cecum, and sometimes partly to its inner side, its tip almost touching the liver or the gall-bladder. In not a few instances it dips downward, passing over the brim of the pelvis. There is no adjacent organ to which it may not become adherent, and in rare instances it is twisted like a loop around the small gut, causing constriction or even strangulation. Osler mentions one case in which the appendix, with the cecum, entered the inguinal canal, curved upon itself, re-entered the abdomen, and was adherent to the wall of an abscess-cavity just to the right of the promontory of the sacrum.

**Pathology.**—Three pathologic varieties are recognized:

(1) **Catarrhal or Obliterative Appendicitis.**—This may be acute or chronic. The term "catarrhal inflammation" is still retained, though scarcely applicable, since, as a rule, appendicular inflammation tends to spread quickly to all the coats, including the serosa. Obliterative ap-

<sup>1</sup> The following names will long be connected with this disease: Pepper, Fitz, McBurney, Porter, Willard Parker, Weir, Sand, Bull, Warren, Keen, Morton, Price, J. William White, Deaver, Senn, and many others. <sup>2</sup> *Sejourn's Annual*, vol. i., 1893.

<sup>3</sup> "Some Points regarding the Appendix Vermiformis," *American Journal of Medical Sciences*, Jan., 1891.



pendicitis is descriptive and in every way preferable. The *mechanism* of the inflammation is briefly as follows: The mesentery being too short, the exit is too small, and in consequence of swelling of the coats (especially the mucous) the venous return is greatly impeded, then the arterial, followed often by abscess-formation. In the female a branch is supposed to be furnished by the ovarian artery, making a more perfect blood-supply. The appearances are, in the beginning, identical with those of catarrhal inflammations elsewhere in the bowel. Within twenty-four hours all the layers are swollen, with marked cellular infiltration, causing the appendix to become firm and often rigid. The mucosa may be denuded of its epithelium and present a granular surface. The external coat (serosa) is usually hyperemic, and not uncommonly the seat of fresh or old adhesions. The tube may become completely obliterated by pressure, resulting in a union between the granular surfaces, in this manner rendering subsequent attacks impossible (Hawkins). It is in cases in which this fortunate result is not reached, however, that acute appendicitis leads to the chronic form with relapses. Two additional terminations may be observed: First, an obliteration of the lumen may occur near the valve, in which case the appendix becomes dilated, and sometimes enormously so (cystic). The contained liquid may be either serous or purulent. Second, obliterative appendicitis may lead directly to ulceration of the mucous membrane, and often in the absence of a fecal concretion or foreign body. Again, the cystic appendix may ulcerate, with or without perforation. Obviously, the more marked the stenosis of the appendix the less favorable the conditions for natural drainage, and the greater the liability to recurrences of attacks of appendicitis. This variety then may end in resolution, complete obliteration, stenosis, or ulceration, and the latter sometimes in perforation.

(2) **Ulcerative Inflammation.**—Like the preceding, this variety may be acute or chronic. It may be a sequel of the obliterative form, and often accompanies chronic obliterative appendicitis. More commonly, however, it is seen in connection with concretions, and sometimes with foreign bodies also. By no means invariably, however, does the presence of these substances excite ulceration of the appendix. Micro-organisms play an important rôle in this variety (*vide* Etiology). The submucosa or muscularis usually forms the base of the ulcer. The termination may be in healing, with tendency to stricture. When obliteration is complete, dilatation beyond the seat of the latter may ensue. Again, the ulcer may extend in depth until perforation occurs.

(3) **Interstitial or Parietal Inflammation.**—This may be preceded by the obliterative or the ulcerative form, which may be followed by anemic necrosis and sloughing. Concretions or foreign bodies are often found, though specific bacteria are of greater etiologic importance. The gravest, most common, and hence the most important lesions are the gangrenous, which are usually limited to a circumscribed part of the tube. Interstitial inflammation has a single termination—perforation—and leads to appendicular peritonitis of a virulent and infectious type.

It may be that neither necrosis nor gangrene may supervene. When perforation occurs, one or more openings, ranging in size from one to several millimeters, may be observed, while the remainder of the appendix may present no abnormalities; more often, however, it is blood-

injected and swollen. The appendix may slough *en masse*. The histopathologic changes may be characterized by intense cellular exudation, necrosis, or purulent inflammation. The muscular coat is hypertrophied; the arteries show obliterating endarteritis.

**Consequences of Perforation.**—A common result of all forms of appendicitis is a localized peritonitis, and this is a constant effect of the severer forms, either leading to (*a*) circumscribed peritonitis or to an (*b*) acute diffuse peritonitis.

(*a*) *Circumscribed Peritonitis.*—At first the surface of the peritoneum is opaque and velvety. Soon a fibrinous exudation covers the appendicular peritoneum, and quickly establishes adhesions between the appendix and the adjacent parts (abdominal wall, intestinal coils). The process may not proceed any further. Generally, however, it is soon followed by a serous or sero-fibrinous exudation, which becomes sero- or fibrino-purulent, and often forms the so-called perityphlitic abscess. The seat of the abscess is always near the tube, and is as varying as the position of the appendix; its size is also extremely variable, as it sometimes contains enormous amounts of pus. Among the most common locations are—McBurney's point, the vicinity of the cecum, the coils of the small intestines (near the umbilicus), and, more rarely, in the pelvis below. The pus contained in the abscess is rarely thick, grayish-yellow in color, and emits a fecal odor; more commonly it is thin, turbid, dark-gray or greenish in color, and has an extremely fetid or even gangrenous odor. The process of gangrenous sphacelation *en masse* is often completed after the limiting wall of adhesion has formed, when the entire appendix is found free in the pus-cavity.

The abscess may be *subperitoneal*, as when perforation occurs into the retro-cecal connective tissue, and the term "iliac abscess" was formerly applied to these extra-peritoneal purulent collections. They are rare, however, since the early operation has been employed. Their situation and dimensions depend upon the direction taken by the appendix. The latter may pass downward, and the pus is then apt to accumulate in the lower part of the iliac fossa, and may point and finally burst in the neighborhood of Poupart's ligament, with subsequent recovery. Occasionally under these circumstances a fistula remains for an indefinite period of time. The appendix may touch various abdominal structures, and the pus in following the line of least resistance may cause spontaneous rupture into the rectum, bladder, or the vagina when it points inward; and into the perinephric region or into the pleural cavity (through the diaphragm) when it points upward; or even into the cecum or colon. The contents of the abscess may also find their way through the abdominal wall in the vicinity of the umbilicus. The psoas muscle may conduct the abscess downward, and it may then point at the hip-joint or gain the gluteal regions or the scrotum, producing the so-called "scrotal appendicitis." The appendix has also been found in a hernial sac. Among the rare lesions to be noted are erosion of one of the arteries of the iliac region (causing fatal hemorrhage) and pyelophlebitis. From the thrombi in the mesenteric veins in the latter condition infectious emboli may be conveyed to the liver, giving rise to hepatic abscess; this occurred in a case of my own at the Episcopal Hospital, Philadelphia. The abscess may also be due

to an extension of the thrombo-phlebitis of the mesenteric veins that lead from the appendix to the portal vein. Thrombosis of the iliac veins with edema of the corresponding leg may also arise, and these veins may, during the process of healing, become compressed, with a resulting edema of the leg, as I have witnessed in two cases. It rarely happens that suppurative processes are both extra- and intra-peritoneal.

(b) *Acute Diffuse Peritonitis*.—This follows perforation when previous adhesions have not taken place or when, having formed, they yield. Generalized peritonitis may also follow the circumscribed form, the lesions being propagated to the entire membrane by direct extension. The morbid changes are those mentioned in the description of Acute Peritonitis. Since the early operation has been employed peritonitis has been the result, usually, of direct perforation before a limiting wall of adhesion has been formed.

**Etiology.**—**Predisposing Causes.**—(a) Doubtless there are *congenital structural defects* that aid in the production of appendicitis. Among them are unnatural length, location, and arrangement of the organ, and peculiarities in the development of its mesentery. These factors tend to obliterate the lumen of the canal by producing kinks and twists, thus favoring the collection of material within the appendix. (b) *Strictures*, particularly near the cecal end of the tube, and adhesions due to old inflammation, especially peritonitis, operate in the same manner as the preceding, only with greater power. (c) *Fecal concretions* are the main cause in nearly one half, while *foreign bodies* play a small rôle, having been present in 7 per cent. only of 1400 cases (J. F. Mitchell). The calculi form in the appendix itself (Rochaz). The foreign bodies are very various, and consist of seeds, worms, gall-stones, pills, bristles, and, more rarely, pointed bodies, as fish-bones or pins. The presence of fecal concretions and foreign bodies is often tolerated by the appendix without symptoms or local pathologic changes; hence they are looked upon rather as a predisposing than as an exciting cause of appendicitis. (d) *Ulcers* (tuberculous, typhoid, and, rarely, actinomycotic) may also produce this affection. (e) *Straining Efforts and Traumatism*.—Not uncommonly excessive muscular exertion, traumatism, or jarring of the body as in jumping, act as favoring causes. (f) *Age*.—The disease is especially frequent in young adults between the fifteenth and thirtieth years. It is not very infrequent in childhood, however, after the third year, and it has even been seen in persons over seventy years of age. (g) *Sex*.—Appendicitis attacks males oftener than females; this fact has been explained (*vide supra*). In the female it is rarely of adnexal origin. Adhesions between the tube and ovary and the appendix may occur, the morbid process then extending to the latter. (h) *Gastro-intestinal Disturbance*.—Indiscretions in the diet may precede a primary attack, and are of paramount etiologic importance in the recurrent forms of the malady. (i) *Heredity*.—That this plays no mean rôle in many cases of appendicitis I have long felt convinced. This serves as the explanation of those cases in which rheumatism and uric-acidemia seem to act as causal agents. (j) Evidence to show that influenza and other affections may cause appendicitis is not wanting. (k) It is not improb-



able that *poor blood-supply* is, after all, the leading predisposing factor, and torsion and the like the active cause.

**Bacteriology.**—While it is true that in many instances there is no apparent exciting cause, yet there are excellent grounds for ascribing specific pathogenic properties to certain micro-organisms. The combined results of several experimentalists tend to show that no special organism plays an exclusive rôle in this disease, but the studies of Hodenpyl indicate that the *bacillus coli communis* is the bacterium most generally present: it is well known, moreover, that this bacillus becomes pathogenic when it escapes into tissues in which it does not naturally belong. Barbaei emphasizes the etiologic importance of the passage of the intestinal contents into the peritoneal cavity—*i. e.* the chemical factor. Of other specific bacteria, those of *typhoid* and *tuberculosis* are not uncommonly found to be present. The *streptococcus pyogenes* may also be found to produce the most virulent infection, and the *staphylococcus pyogenes aureus*, the *proteus*, and other specific organisms have been found. The great frequency of appendicitis is rendered appreciable by the numerous favoring factors (including the congenital conditions) acting upon the appendix, which naturally has an exceedingly low vitality; also by the constant presence of one or more organisms that are known to become pathogenic in the presence of a slight lesion.

**Clinical History.**—Doubtless many cases are overlooked because of the extreme mildness of the symptoms. These are often attributed to intestinal indigestion or to a “cold,” to which the patient pays little attention unless he displays unusual susceptibility.

The *onset* of acute appendicitis may be slow and gradual, but oftener it is *quite sudden*. A clear history of some obvious cause (an error in diet or muscular effort) may be obtainable. Again, preceding the onset of the definite symptoms and extending over a day or two, there may have been *certain prodromes*, as impaired appetite, nausea, constipation, or diarrhea. In slow cases the local and general symptoms are at first slight, but gradually increase in severity as the different stages of the disease are evolved. Indeed, in the latter class the patient may go about his customary duties during the attack with ill-defined rational symptoms, while in reality suffering from periappendicular abscess. These patients run two serious dangers—first, spontaneous rupture of the abscess into the peritoneal cavity may occur; and secondly, the slow septic absorption may suddenly overwhelm the system. As a rule, the sudden cases develop in seeming perfect health, and are sometimes heralded by a rigor or chilliness.

The characteristic features of the invasion are *abdominal pain, fever, tenderness over McBurney's point, circumscribed resistance, gastric disturbances*, and, as a rule, constipation. The *pain* varies in intensity from a mere feeling of soreness to that of the most agonizing suffering. It may be paroxysmal, though oftener it is constant, with moderate exacerbations. Severe pain points to an involvement of the peritoneum and signalizes a danger of perforation. At first the pain may be referred to any point in the abdomen; later it becomes more distinctly localized in the ileo-cecal region.

*Elevation of Temperature.*—The exacerbations may at first touch



sions above and in front of the anterior iliae spine. The position of the indurated area varies according to the location of the appendix, but is usually found at or in the vicinity of McBurney's point. Sometimes a resistant mass of the shape and size of an enlarged appendix is palpable. In such cases peritoneal exudation has not as yet occurred to any great extent. In some cases the induration is diffuse at first, but assumes the usual circumscribed form later; it may, moreover, be so deeply seated as not to be appreciable. The degree of tenseness of the two recti muscles—right and left—should be compared, though an absence of tension of the right rectus does not, I feel certain, eliminate the possibility of appendicitis. The results of *percussion* furnish no certain guide. As a rule, the note on light percussion differs from that on the opposite side; on deep percussion a dull tympany or a circumscribed area of dullness can be outlined. This deadness may be due in great part to the presence of fecal matter in the adjacent coils of intestine. While at the start the abdomen may be flattened or even retracted, tympanitic distention afterward appears, particularly in the cecal region, giving rise to exaggerated tympany on percussion.

Less characteristic, though still of diagnostic worth, are certain other symptoms. At the beginning *vomiting* usually occurs, unless there be diarrhea, and is attended by more or less nausea; it may continue throughout the course of the attack. In most cases, however, after a few fits of vomiting the symptom disappears, though it may recur if errors in diet be committed or if peritonitis supervene. During the attack constipation is the rule, though diarrhea, which sometimes precedes appendicitis, may also occur at a late stage as a septic symptom. There is anorexia, and the tongue is coated. The *decubitus* is dorsal, with the right leg flexed. Frequent micturition (early) and retention of urine (later) are not uncommon, the urine having a deep color-tint, and sometimes containing albumin.

The case may follow a mild *course*, terminating in resolution with recovery; or it may be of a severe type and develop perforation, with the formation of abscess or diffuse peritonitis. As graphically stated by Fitz, it is impossible to obtain statistical evidence on a large scale of the relative frequency of these alternatives, and hence the frequency of treatment of appendicitis by abdominal section. From all available data, however, it would appear that in more than one-half of the cases the course is light and favorable.

If not operated upon early, the fever may continue for three to five days, and then subside, with simultaneous abatement of the severe local and general symptoms and with the establishment of convalescence. The same amelioration of the symptoms may be brought about by early free purgation, either as the result of salines or, rarely, spontaneously. In these instances resolution takes place even after invasion of the peritoneum. Small abscesses may be absorbed, and usually in cases terminating in resolution perforation has not occurred. Infection of the peritoneal membrane directly through the appendix is not uncommon.

*In severe attacks perforation may occur, with the development of localized peritoneal abscess or generalized peritonitis (vide Pathology), and it must be remembered that cases that begin gradually may also show a tendency toward perforation. When this event occurs early in*



the course of a severe attack or after a protracted mild appendicitis the symptoms of *local* or *general peritonitis* are superadded. If early, the symptoms pointing to peritonitis are intense; the abdomen swells quickly, and is exquisitely tender, though the physical signs of a tumor are absent. The temperature often falls, and the characteristic vomiting and circulatory collapse appear. Often the generalization of the peritonitis is marked by less violent symptoms. Starting from the seat of circumscribed inflammation, the pain and tenderness propagate themselves noticeably from day to day until every portion of the peritoneum has been invaded. Besides progressive augmentation in the local features, including the pain, there is a gradual failure in cardiac power, as shown by the condition of the pulse; vomiting also returns, and at last becomes fecal. Death results from asthenia, and sometimes suddenly when unanticipated. If perforation occurs later, sufficient time has been allowed usually for the inflammation to become circumscribed, in which case the localized abscess is generally intra-peritoneal; it may, however, rarely be extra-peritoneal. The local symptoms intensify, the pain becomes excruciating, and the spot of tenderness may rapidly extend itself in all directions, particularly downward. Vomiting sets in, and may become troublesome, and constipation is absolute, not even gas escaping from the rectum. Retention of urine is common.

**Physical Signs of Localized Abscess.**—*Inspection* shows distention of the belly, the affected area being prominent, with an obliteration of the natural depression in the right iliac region. *Palpation* discovers induration and great tension that soon yield to pressure (doughy), and edema of the skin. If the abscess is superficially seated, fluctuation may be appreciated on bimanual palpation. Deep-seated tumors are not uncommon, and then fluctuation is detected with difficulty or not at all. An examination *per rectum*, with a view to determining whether the abscess has gained the pelvis, is highly important. Counter-pressure above with the free hand aids materially. In doubtful cases bimanual pelvic examination should not be neglected. *Percussion* reveals dullness if the abscess be superficial. A tympanitic note, however, is often elicited, due either to an intervening coil of intestine or to the gas contained in the sac of the abscess.

If active peritonitis and septicemia do not develop, the constitutional as well as the local symptoms may abate, and the patient leave his bed, carrying with him, however, the abscess. The latter may point somewhere in the right lower quadrant of the abdomen or in the lumbar region. There is also a strong tendency toward spontaneous rupture into the rectum, bladder, vagina, or cecum. Often, preceding the discharge of pus into these organs, the latter display marked irritability, particularly the rectum and bladder. There is always the danger that the contents of the abscess may find their way into the general peritoneal cavity. The symptoms of hepatic abscess may develop at an advanced stage. The pus may traverse the abdomen in the upward direction until it touches the diaphragm, when the symptoms of subphrenic abscess may be manifested. Extension through the diaphragm may now occur, causing pleurisy or pericarditis, and a pleuro-fecal fistula may thus be established.

The general symptoms undergo a modification, due to the *suppurative*

*process.* Rigors or a decided chilliness may occur. Diarrhea often succeeds to previous constipation, and drenching sweats to a dry skin. Improvement and even spontaneous cure may ensue if spontaneous rupture into one of the outlets of the body should occur. The fever (Fig.



FIG. 59.—Temperature-chart of a case of appendicitis. R. C.—, aged nineteen years; carriage-builder. A peritoneal abscess was found, while the appendix was becoming gangrenous.

59) may be either remittent or intermittent, and if the localized inflammatory process be active, the usual pronounced features of septicemia are predominant in the clinical picture. The latter specially grave condition often drifts into an extreme typhoid state with a hopeless course.

**Diagnosis.**—Typical cases of appendicitis are readily diagnosed. Their recognition rests upon a few cardinal symptoms—viz. the acute development of severe pain in the right iliac fossa, coming on in a person previously healthy and usually under forty years of age; appendicular tenderness, unilateral induration, fever, vomiting, and constipation, or, more rarely, diarrhea. Atypical cases, however, may offer difficulty, although Morris affirms that errors in diagnosis are less frequent than in almost any other disease. Often the pain is, for a time, referred to a circumscribed area far removed from the usual site of the appendix, and rarely it continues without a change of situation throughout the attack. In the latter case the local lesions may occupy the usual, though oftener they have an unusual, position. Thus, when the pain is referred “due east,” or to the left iliac fossa, with

bilateral induration, the appendix will be found in the pelvis (Deaver). In such instances a rectal and a bimanual vaginal examination are imperative. It should be an unvarying rule in all cases of severe abdominal pain to palpate with the finger-tip every square inch of the abdomen if necessary, to find the localized tenderness when it is not found at McBurney's point. The degree of tenderness sustains a close relationship to the severity of the local inflammation as long as the condition remains strictly localized, but this relationship is lost when generalization occurs. With the appearance of a circumscribed induration and of the intense local tenderness and pain it is reasonably sure that perforation either has occurred or is impending. Perforation, however, may occur without local induration, and even after subsidence of the acute pain and excessive tenderness. Gangrenous appendicitis is *most deceptive*. The very acute symptoms, including the fever, may disappear, and unless the physician be upon his guard the patient will be considered convalescent and be allowed to go about. Rupture of the abscess now occurs unexpectedly into the peritoneal cavity, intestines, or some other direction, or a large-sized abscess develops with the usual signs and symptoms.

**Differential Diagnosis.**—*Typhlitis, and especially the Massing of Feces in the Cecum.*—These are truly rare conditions. According to McBurney, 99 per cent. of all typhlitic abscesses are of appendicular origin, and of 400 autopsies by Einhorn 91 per cent. had this origin. Ball and others have performed laparotomy for ulcerative cecitis, but this condition cannot be recognized during life. Stercoral typhlitis is discriminated from true appendicitis by the precedent constipation, which may become absolute, by the dragging character of the pain, the late-appearing fever, and chiefly by the physical signs, which indicate the presence of a superficial, sausage-shaped tumor that is often doughy and extends vertically from a point near the right costal border "southward" through the ileo-cecal region. Percussion elicits dullness over the seat of the tumor. The localized tenderness and circumscribed resistance of acute appendicitis are wanting, and a thorough emptying of the large intestine usually cures stercoral typhlitis.<sup>1</sup>

*Renal Colic.*—The absence of fever and of a localized spot of tenderness and induration, and the presence of hematuria are points that distinguish this affection from appendicitis.

*Indigestion.*—Digestive disturbances, and particularly pain and vomiting, accompany appendicitis. When they occur independently of appendicitis, however, they can be relieved, and the appendicular region remains free from fixed pain, tenderness, or tumor.

*Acute Inflammation of the Gall-bladder, with Distention.*—This gives rise to a superficial, mobile, pear-shaped tumor, with or without jaundice—features not met with in appendicitis. Osler, however, mentions a case of the sort in which the diagnosis was undetermined until laparotomy was performed.

*Perinephric Abscess.*—Without a history indicative of chronic renal disease or of nephro-lithiasis the differentiation cannot be made except by exploratory incision.

<sup>1</sup> It is highly probable that the term "stercoral typhlitis" is synonymous with chronic appendicitis with retained feces in the cecum.



*Carcinoma of the Large Intestine.*—This discriminates itself by its peculiar and more chronic history.

*Acute Peritonitis, due to Ovarian or Tubal Disease.*—When the appendix occupies, not its usual seat in the iliac region, but the pelvic fossa, then the distinctions between salpingitis and appendicitis are not easily drawn. Right ovaritis, owing to the presence of pain, tenderness in the right iliac fossa, and fever, often closely simulates appendicitis. In the former tenderness is less pronounced, and the organs of uterogestation manifest certain disturbances of function. A clear history, coupled with a careful pelvic examination, will usually complete the clinical separation of these two conditions.

*Extra-uterine Pregnancy.*—In this condition the menstrual history furnishes important information. There is, in addition, profound collapse, due to hemorrhage, when rupture of the adhesions occurs. Elevation of temperature is absent. The localized tenderness and increased resistance are lower in the pelvis than in appendicitis.

*Acute Tuberculous Peritonitis.*—As in appendicitis, so in tuberculous peritonitis, pain, tenderness, and fever are present, but in the latter the onset is more gradual, and the signs of tumor and increased resistance in the right iliac fossa are absent. Movable dulness may be present in the tuberculous affection, and not in appendicitis until the peritonitis has become generalized. The lungs are generally implicated in tuberculous peritonitis.

*Acute Intestinal Obstruction.*—When this is due to intussusception there may be signs of a tumor, but not at McBurney's point; the tenderness over the site of the mass is less intense, while the frequent bloody discharges that are seen in this condition, accompanied by tenesmus, do not characterize appendicitis. When obstruction is caused by strangulation stercoraceous vomiting is apt to occur, and is absent in appendicitis. Pain, local tenderness, and, not uncommonly, signs of a tumor appear, but elsewhere than at McBurney's point. Some of these instances, however, remain obscure till the diagnosis is set at rest by the celiotomist.

*Hip-joint Disease.*—In both hip-joint disease and appendicitis the dorsal decubitus with flexed leg is noted. If the patient be anesthetized, however, full extension of the leg and a normal condition of the hip-joint are easily demonstrable in appendicitis.

*Typhoid Fever.*—Mild cases of appendicitis with accompanying diarrhea bear a close superficial resemblance to typhoid fever. In the latter affection, however, the onset is more gradual and the fever-type more continuous than in appendicitis. In typhoid the stools are somewhat peculiar, the spleen is swollen, there is dulness of intellect, bronchitis and the characteristic eruption attend,—all features that are absent in appendicitis. The diazo-reaction, if present, would strengthen the diagnosis of typhoid, and a response to Widal's test would be conclusive. In appendicitis the local features, and in typhoid the general, are predominant.

*Dietl's Crises.*—In a case of movable kidney which I saw recently all the symptoms pointed to appendicitis. An operation was about to be performed when a sudden subsidence in the abdominal swelling and local induration occurred. The kidney was subsequently detected in an abnormal location (*vide* Mobility of the Kidney).

## CHRONIC APPENDICITIS.

*(Relapsing Appendicitis.)*

Relapses occur in nearly one-half the total number of persons who have suffered from a primary attack of appendicitis. In most of these cases there is constantly present a slight local discomfort during the interval; in a small percentage, however, there is an entire freedom from uneasiness. The *local symptoms* in those having had an antecedent peritonitis are more pronounced than in the first attack, but after a number of recurrences the symptoms are likely to be less severe with each new attack. The most constant symptom between attacks is a sub-acute form of *pain* that is liable to manifest exacerbating periods with slight fever. Physical fatigue, a strain, and errors in diet causing gastro-intestinal disorder are very likely to induce a relapsing appendicitis. Chronic appendicitis strongly favors the retention of fecal matter in the cecum, thus forming so-called *stercoral typhlitis*. This association was formerly mistaken for primary typhlitis. The characteristics on which the diagnosis is based during the attack are similar to those detailed under Acute Appendicitis; the course is, however, somewhat more condensed than that of the acute form.

In the intervals between the attacks the appendix can be readily appreciated on *palpation*, the method employed by Edebohls being preferable: "The patient lies upon his back with the examiner at his side; the latter places his right hand upon the patient's abdomen over the right rectus muscle, opposite the anterior superior spine of the ilium, and presses the left hand upon the right, so that no force is used by the right hand and the tactile sense of its fingers is left undisturbed. The hands are drawn slowly outward, allowing the contents of the abdomen to slip from underneath them. The coils of intestine can be felt as they escape from under the hand as it presses against the posterior abdominal wall."<sup>1</sup> In this way the appendix may be felt as an elongated tumor of the size and shape of the little finger. If there be only a slight exudation present, the appendix often appears to be immediately beneath the abdominal wall. It may, however, be deep-seated, even though the exudation with adhesions be absent. Both pain and tenderness are pronounced, and particularly if pus be present.

The results of chronic appendicitis upon the general health and nutrition of the patient are quite noticeable, and tend to augment as time passes, if the attacks be frequent or the intervals between them grow shorter. The chief symptoms are those of a nervous type; emaciation and debility are also observed. The associated nervous symptoms are those of neurasthenia. These patients often become introspective and exceedingly irritable, the mental condition being accounted for, to a great extent, by the consciousness that there is ever present the overhanging danger of a fresh attack with serious possibilities.

**Differential Diagnosis.**—*Carcinoma of the Cecum.*—This presents many points of similarity to chronic appendicitis. I have under my care at present a lady aged sixty years suffering from chronic appendicitis, whose case had been diagnosticated as carcinoma of the cecum, and for a considerable time my own view coincided with that of my predecessor.

<sup>1</sup> B. Farquhar Curtis: *Twentieth Century Practice of Medicine*, vol. viii.

The occurrence from time to time, however, of relapses, during which the feces were massed in the cecum and fever arose, soon indicated the correct diagnosis. Besides the absence of periodic attacks of fever, the general features—loss of flesh and strength, anemia—are more steadily and rapidly progressive in carcinoma of the cecum. The history of the mode of onset also aids in the distinction. Pain, tenderness, and a resistant tumor are common to both affections.

*Hypochondriasis and Hysteria.*—Hypochondriasis and hysteria may lead to the manifestation of morbid feelings simulating those of appendicitis. Such cases may show merely a greatly exaggerated uneasiness, or such an increase of sensibility as to cause the patient to complain of pain in the right iliac fossa. In addition, there may be localized tenderness. I recently witnessed the removal of the normal appendix from an hysteric female in whose family two genuine cases of appendicitis had occurred not long previously. Hypochondriasis and hysteria distinguish themselves by the antecedent history and by the absence of a tumor-mass and of increased resistance; there is also an absence of localized tenderness if the patient's attention be withdrawn. In such subjects oxaluria is not infrequent, and it is possible that irritation of the right ureter by the passage of crystals of calcic oxalate, as mentioned by Cabot, may explain the localizing of the discomfort (Wood and Fitz<sup>1</sup>). I recently saw a case of this sort in a neurasthenic medical student.

#### RECURRENT APPENDICITIS.

When successive attacks occur in the same individual at intervals varying from several months to a year or more, each new attack is spoken of as a *recurrent appendicitis*. Severe attacks may succeed light ones, or, conversely, mild recurrent may follow severe preceding attacks. I recall several cases in which rudimentary appendicitis (indicated merely by colicky pain) occurred, and lasted from a few hours to a day or two. Often the illness is too trivial to lead the patient to consult a doctor. An absolute diagnosis demands, besides the subjective symptom, pain, the presence of localized tenderness (with or without induration), and elevation of the temperature. In several subjects of recurrent appendicitis formerly under my care the last attack occurred three or four years ago. That each new attack may be the last is always to be remembered.

**Prognosis.**—In forming the prognosis in a given case of appendicitis the same rules may be followed as in the case of acute infectious diseases. To estimate the severity of the type of infection, however, is not a simple matter. Unlike many of the acute infectious diseases, the height of the temperature and, to a lesser degree, the rate of the pulse are unreliable guides in appendicitis. Broadly speaking, however, in the severer forms the local process exhibits a strong tendency to spread; the temperature and pulse are relatively high, and there is an intense appendicular intoxication. These are the cases that suppurate or result in perforative peritonitis (often rapidly spreading), and in pericecal abscesses. They are among the gravest of known conditions. Of this fatal group of cases not less than 68 per cent. die before the eighth day.

<sup>1</sup> *The Practice of Medicine*, p. 886.



The development of *fulminant peritonitis* or of a peritoneal abscess after perforation is attended by a falling temperature, though subsequently the latter may mount high or become markedly irregular.

On the other hand, in the mild forms that are included in the name *catarrhal appendicitis* recovery is the unvarying rule. These lighter cases often lead to adhesive peritonitis—a circumstance that strengthens the view that they are of an infectious nature. The temperature is only moderately elevated as a rule, and the pulse-rate correspondingly quickened. Both pulse and temperature indicate marked improvement on the third or fourth day, while the pain and localized tenderness disappear. In this connection the deceptiveness of gangrenous cases must be recollected (*vide supra*, Diagnosis). The complications that are most likely to arise and other points of prognostic significance have been fully stated in the Clinical History. The general mortality of appendicitis is about 14 per cent. (Fitz). Improved methods, chiefly surgical, of dealing with the disease have, however, greatly reduced its death-rate. The prognosis in *chronic appendicitis* is most uncertain; after the patient has survived several attacks it is on the whole more favorable.

**Treatment of Appendicitis.**—Whether imminent danger of perforation exists or not, the physician who is called to a case of appendicitis should at once request the services of a competent surgeon. Few surgeons subscribe to the doctrine that all cases demand operation; but, since it may become necessary to perform celiotomy at any hour thereafter, the latter should help to settle the important question: “When is it necessary to operate in the case?” The physician who does not pursue the course above recommended falls short of his duty, both toward the patient and toward the surgeon on whose skill he relies to remove safely the source of danger. Surely, in a disease that so often baffles both physician and surgeon, suddenly developing, as it sometimes does, a fatal virulence without previous unfavorable symptoms, they should stand guard together from the moment the case is diagnosticated or appendicitis is strongly suspected. Unfortunately, both the medical and surgical treatment of appendicitis have recently been recommended with great earnestness by their respective advocates.

With rare exceptions, prompt surgical intervention should be recommended. The indication for an immediate operation is undoubted in all cases of acute appendicitis, whether marked by sudden and severe or mild invasion-symptoms, if seen at the beginning of the attack, and free purgation at the earliest possible moment is not followed by decided relief. A waiting policy and medical treatment are also perilous in doubtful cases. Obviously, the conditions are less favorable for operation after a case has progressed to the beginning of abscess-formation—*i. e.* from the third to the fifth day of the illness. It is at this period that the peritoneal inflammation tends to circumscribe itself by the formation of adhesions. Hence, as Richardson states, it is “too late for an early operation, and too early for a safe late operation,” since there is great risk of infecting the general peritoneal cavity. Whether it is wise to allow the appendix to remain after adhesions have been formed in some cases, and merely to drain, cleanse,

and pack the cavity, cannot be discussed here. The mild attacks that develop in the course of chronic appendicitis after numerous previous seizures need not excite alarm. Under such circumstances operation should be undertaken between attacks, when the mortality is practically *nil*. On the other hand, in cases that have been allowed to drag on until general peritonitis has set in, treatment by operation is not advisable. Moreover, the most ardent advocate of immediate operative treatment is sometimes compelled to rest satisfied with medical measures. Such cases are those in which there are associated chronic affections (advanced diabetes, Bright's disease), not to speak of those in which the patient declines operation. Hence there is a medical treatment of appendicitis, but it should not be the treatment of election.

**General Management.**—The patient should be kept in bed in a quiet, well-ventilated apartment, and in no affection is the value of *absolute rest* in the treatment of inflammation greater than in appendicitis. The diet should be liquid and nutritious, consisting chiefly of pancreatized milk and concentrated broths. All articles of food that tend to undergo fermentative changes, and all carbonated drinks, should be prohibited, since they increase meteorism. The patient should be under- rather than over-fed. At the start, and particularly if a sausage-shaped tumor be present, intestinal irrigation, oft-repeated with a view to removing the fecal matter, must be carried forward assiduously. Saline laxatives (Rochelle salts, ʒij—8.0—every hour or two, preceded by a dose of castor oil or a few fractional doses of ealomel) are to be administered until the evidence of their action upon the bowels has been definitely noted. There almost never exists a contraindication to the use of *saline aperients* at the onset of the attack, and they constitute the best known means of obviating, as well as limiting, the spread of peritonitis by depleting the portal system and emptying the bowels. If commenced early, they may be continued throughout in doses sufficient to produce two or more daily evacuations. In the event of a development of evidences of peritonitis with pus-formation, salines should be pushed vigorously, unless an operation can be promptly performed. I am aware that many authors advocate withholding purgatives when indications of suppuration appear, but I have yet to see a case in which perforation has followed an active saline treatment. I avoid the use of high enemata in progressive cases, since they are more apt than salines to induce rupture of the sac.

As regards the use of opium professional opinion is not united, though a general tendency toward the limitation of its use to the minimum amount necessary to alleviate pain is happily noticeable; unless demanded by excessive suffering it had better be omitted altogether. When necessary, it is best administered hypodermically in the form of morphin (gr.  $\frac{1}{12}$ — $\frac{1}{8}$ —0.0054—0.0081). The greatest objection to the use of opium is its effect in veiling the symptoms that assist the physician in forming a judgment as to the prospects and progress of the case.

**Local Measures.**—The suspended ice-bag is an excellent means of combating the pain, and often obviates the necessity of an internal use of opium. Instead of the ice-bag, cloths wet in cold water may be applied and changed every few minutes. In the early stage a few leeches may be beneficial in their effect upon the local inflammation. Blisters, however, are rarely advisable, and are particularly objection-

able should the patient afterward be submitted to an operation. Mild forms of counter-irritants (mustard-paste) are preferable, though these also render the skin and underlying tissues hard and leathery.

**Management of Convalescence.**—The patient should not be allowed to leave his bed for several days after the disappearance of all symptoms; even the mildest forms of exercise should not be undertaken for at least one week subsequent to getting out of bed. During convalescence the diet must be carefully guarded, and the bowels, at all hazards, kept in a soluble condition. It is questionable whether drugs will aid in the absorption of the exudate or assist in resolution. Gentle and persistent counter-irritation with preparations of iodine will be found useful.

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## INTESTINAL OBSTRUCTION.

(*Ileus.*)

**Definition.**—An acute or chronic, complete or partial, occlusion of the intestinal canal.

**Pathology and Etiology.**—The causes of intestinal obstruction may be divided, at once most simply and practically, into the (1) *acute* and (2) *chronic* forms. In the former variety the narrowing or closure develops very suddenly or rapidly, and usually in the small bowel; in the latter, the large bowel is commonly affected by pathologic conditions that develop slowly and gradually and narrow its lumen; the latter conditions usually occur in persons of advanced years.

**Acute.**—(a) *Strangulation.*—In the order of frequency, this is first among the causes of acute intestinal obstruction. It is produced most often by bands of adhesion, the result of a former recent or remote peritonitis, and is most commonly situated in the right iliac fossa. Incarceration of the bowel from flexions and adhesions not rarely follows upon abdominal section for the treatment of pelvic disease in women.

The usually free end of Meckel's diverticulum is sometimes attached to the abdominal wall, and may thus cause constriction of a loop of bowel. This diverticulum is the remains of the fetal omphalo-mesenteric duct, and arises from the ileum about half a meter (1.64 ft.) from the ileo-cecal valve. A similar constricting band is formed by a cord representing one or more of the obliterated omphalo-mesenteric vessels. The adhesive attachment of the free end of the appendix vermiformis may also form an opening through which the bowel may be caught.

Internal strangulation (hernia) may be the result of forcing a portion of bowel through a slit in the omentum or mesentery, or into peritoneal diverticula and openings, such as the duodeno-jejunal fossa (*Freitz's retro-peritoneal hernia*) or the foramen of Winslow.

Diaphragmatic herniæ are not of extreme rarity, and may be either of congenital or traumatic origin. Most cases of intestinal strangulation occur in males during early adult life.

(b) *Intussusception.*—*Invagination* is the descending "telescoping of one section of the bowel into another," probably caused by a circumscribed, irregular peristalsis of the intestine. The effect of the latter state in producing invagination may be either a thrusting forward of



the receiving portion by a contraction of the longitudinal muscular coat (Nothnagel), or a thrusting inward and downward of the portion immediately above by means of an increased or spasmodic peristaltic action. Thus, a cylindric or sausage-shaped tumor results, varying from a half inch to over a foot (1.3–30 cm.) in length. The layers met with in intussusception are the outer or receiving, called the *intussusciptions*, the middle or returning layer, and the inner, called the *intussusceptum*. The seat of invagination is most commonly at the ileo-cecal valve, though it is often found in either the ileum or colon alone. Sometimes the intussusception occurs and is detected in the rectum. A lateral or partial invagination, more or less chronic, may also occur, due to the attachment of a tumor within the bowel.

The intussuscepted portion of intestine is usually the seat of peritoneal adhesions and considerable tumefaction, so that in pronounced cases the parts are so firmly agglutinated that reduction is wellnigh impossible. The engorgement may pass into an intense local inflammation, with final necrosis and sloughing, and even the discharge *per rectum* of the invaginated portion; or a fatal termination may be ushered in by perforation of the bowel.

Intussusception occurs most frequently by far in children prior to ten years of age, in whom also the disease is more acute than in adults. Males are more subject to invagination than are females.

Invagination is asserted to be an occasional consequence of the operation of circular enterorrhaphy and of lateral anastomosis by plates (Robinson).<sup>1</sup>

(c) *Volvulus*.—Twists of the intestine are met with most commonly at the sigmoid flexure of the colon. An unusually long or relaxed mesentery predisposes to the condition, so that the axis of twisting may either consist of the mesentery itself or frequently of the bowel. Not rarely the pedicle of the volvulus contains both a twist and a sharp bend in the bowel, causing complete acute strangulation. The latter condition may be pronounced in such cases, or at least be hastened, by the accumulation of the intestinal gas and of masses of feces, or by bowel-adhesions to an adjacent stump of omentum (Nieberding). The passive reactive pressure of the coils of intestine and of the abdominal walls tends also to further confine the enormously dilated and twisted loop of bowel to its abdominal state. Knots may be formed by the association of loops of the ileum with each other or about the pedicle of a twisted cecum.

Here, again, males between forty and sixty years of age have been observed to be especially the subjects of volvulus.

**Chronic.**—(a) *Fecal Impaction*.—*Intestinal Concretions*.—Accumulation of feces (*coprostasis*) is a common cause of intestinal obstruction, the impaction taking place usually in the cecum or sigmoid flexure.

Though not infrequent in children, fecal obstruction is more common in adults (particularly in females), in the hysteric, the demented, and the hypochondriac. Congenital dilatation of the colon may predispose to coprostasis, and an acquired dilatation, which in some cases becomes enormous, is often the result of paresis of a portion of bowel caused by over-distention for a long period of time. The retained fecal masses may become hard, but for some time permit the passage of soft or liquid

<sup>1</sup> *Med. Record*, Aug. 13, 1892.

material through the interstices of the accumulation, until finally either complete obstruction takes place or the condition is relieved. So severe may the obstruction prove in some cases as to result in inflammation, ulceration, and even perforation of the bowel.

Among other causes of obstruction due to abnormal contents may be mentioned *enteroliths*. These are intestinal concretions formed of various nuclei, as gall-stones, hardened feces, phosphates of lime and magnesia, various foreign substances, and organic derivatives. Balls of tangled ascarides may mass sufficiently to cause obstruction.

Gall-stones not infrequently become impacted in the duodeno-jejunal or ileo-cecal regions after ulceration through the duct, except in the case of very small stones, which enlarge subsequently by accretion.

Foreign bodies, as pins, buttons, coins, fruit-stones, may also cause obstruction of the bowel. It is stated that even insoluble mineral medicines, as bismuth or magnesia, have caused obstruction by accumulation in the intestines.

(b) *Tumors*.—Tumors cause a form of chronic obstruction that may at any time develop suddenly into the acute type. They may do so either as—(1) *new growths* in the wall of the intestine itself, or by (2) *compression and traction from without*. Again, the intestinal neoplasms may be *malignant* or *benign* in nature. *Carcinoma* of the bowel is at once the most frequent and important of these. It may be either circumscribed and annular, causing a gradual narrowing of the bowel-lumen, or a diffused infiltration of the intestinal wall, commencing either in the mucosa or in its glands (cylindric epithelioma). Its most common seat of growth is the large bowel, about the sigmoid flexure. The mesenteric and retroperitoneal glands are usually secondarily affected. Ulceration of the bowel and catarrhal inflammation of the mucous membrane above the carcinoma may coexist late in life.

Sarcoma usually attacks the small bowel, starting beneath the mucosa, and is of the recurrent variety. Regional infection of the mesenteric and retroperitoneal glands (*Löbstein's cancer*) is also a usual consequence of sarcoma. It may occur in children or in young adults.

Benign tumors may be polypoid, adenomatous, fibromatous, and lipomatous. Intestinal obstruction due to compression or traction may be caused by tumors (omental) or by adhesions of the pelvic viscera.

(c) *Cicatricial strictures* cause chronic intestinal obstruction, as after the healing of various ulcers, the cicatrices of which slowly contract. Cicatricial stenosis of the colon is commonly due to the cicatrization of dysenteric ulcers. In the rectum the stenosis is usually a result of a syphilitic lesion. Tuberculous and, very rarely, typhoid ulceration may be followed by stricture of the small intestine.

(d) *Congenital stricture* is rare, and is more purely surgical than the preceding cases. It is often an occlusion or an imperforate condition of the anus (*atresia ani*), and is only mentionable in this connection.

(e) *Paresis of Peristalsis*.—This condition—called also *adynamic obstruction*—while it is a functional affection, is held to be either a circumscribed or diffuse paresis of the intestinal muscular coat. It is caused by some such inflammatory disturbance as enteritis or peritonitis, or even by the manipulations employed in prolonged abdominal sections. In such cases the obstruction is due to an accumulation of feces and

gases in the paretic portion of the bowel, causing marked tympanites, vomiting, and constipation.

**Special Pathology.**—The pathologic changes that accompany nearly every form of intestinal obstruction are briefly stated as follows: Accumulative dilatation—with hypertrophy in chronic cases—of the intestine above the seat of disorder, and an emptiness, narrowing, and even atrophy below the obstruction. The affected walls of the bowel are inflamed, and there is a surrounding acute or chronic peritonitis. Catarrhal and sometimes diphtheritic inflammation of the mucosa may develop. Gangrene, ulceration, and perforation of the bowel, with resulting generalized peritonitis, may also ensue.

**Symptoms.**—**Acute Obstruction.**—There is a suddenly developed *abdominal pain* that may follow some abrupt or severe exertion. *Early vomiting* and *absolute constipation* are also conspicuous and important symptoms. If the obstruction is high in the small bowel, distressing hiccough and eructations may precede the vomiting. Except for the possible discharge of the intestinal contents below the seat of obstruction, the constipation is usually complete and obstinate. Accompanying the latter condition there is tympanites, which is most marked in obstruction of the colon. Intermittent and colicky at first (partial obstruction—Treves), the *pain* soon becomes agonizing and constant. *Vomiting*, also, alternating with *painful retching*, is more constant and severe after several hours. The *material* at first ejected is gastric and mucous; it then becomes bilious, and finally is characteristically stercoraceous, due, most probably, to the putrid decomposition of stagnated contents above the obstruction.

The *constitutional symptoms* develop early, are intensely threatening to life, and cause rapid and profound depression and collapse. The pinched and pallid features, cool and moist skin, Hippocratic expression, rapid and feeble pulse, the usually subnormal temperature, shallow and accelerated breathing, marked thirst, scanty urine, great anxiety and prostration,—all indicate the gravity and danger of the condition.

The *physical examination* will discover a swollen, extremely tender, and tympanitic belly. Exaggerated peristalsis of the intestine above the obstruction may be visible on the surface of the abdomen. Borborygmi, gurgling, and splashing may be heard on auscultation.

**Chronic Obstruction.**—The symptoms are more dependent upon the special causes operating than in acute obstruction. The fact that early in the case only partial obliteration of the intestinal lumen may be rightly inferred in many of the chronic forms of obstruction has given rise to the discriminating term of *intestinal constriction*. In general, the clinical history is one of *increasing* and *intractable constipation*, sometimes alternating with diarrhea, due to catarrhal inflammation of the mucosa above the obstruction. Paroxysms of colicky *pain* and, later, augmenting *tympanites*, *vomiting*, and *prostration*, attend. These symptoms may merge suddenly into those of the acute form of obstruction. The bowel-movements in chronic obstruction are irregular, infrequent, slight, and sometimes accompanied by pain and tenesmus. The *stools* consist often of small, hard, ribbon-like, or scybalous masses, and may contain blood and mucus. When the stenosis is in the small intestine the constipation is less apt to occur on account of the fluidity of the



contents. Sometimes, and particularly in old people, the rectum becomes distended with hardened accumulations of feces; there is in such cases a constant feeling of fulness and a harassing desire to defecate, but the attempts thereat are ineffectual. The pain of fecal impaction may be due either to colitis or to peritonitis, and may be referred to the regions of the cecum or sigmoid flexure.

In *malignant* and in *cicatricial stenosis* there are a prolonged and variable history of constipation, occasional vomiting, localized pain, meteorism, and, in cancerous cases, the development of the characteristic cachexia and the progressive emaciation.

**Physical Examination.**—*Inspection* shows the abdomen to be distended from meteorism, the movements, and contour even, of the coils of intestine in active peristalsis above the seat of stricture being evident. A tumor or the throbbing aorta (excited, perhaps, by pressure of the distended bowel or growth) may be *palpated*. Tympany and borborygmous noises may also be noted.

**Diagnosis.**—**Locality of the Obstruction.**—Given the symptoms of a sudden, severe, and exacerbating pain in the abdomen; of marked, and later feculent, vomiting; of absolute constipation and of tympanites and profound, early, systemic depression,—a diagnosis of acute intestinal obstruction may be easily made. The determination of the seat of trouble, however, is often very difficult. First may be mentioned the differential diagnosis between obstruction occurring in the small and in the large intestine. It may be noted of the former that vomiting occurs early, is scanty, and later feculent, while in the latter there is less vomiting and the vomitus is seldom feculent. Again, in obstruction of the small gut the distention is both less marked and higher situated, while in that of the large gut tympanites is often quite marked, is more central, is associated with tenesmus, and sometimes with mucus and blood. If the cause of obstruction be a tumor or stricture, the locality may be successfully palpated or the lower limit of the active coils of hypertrophied intestine may be defined.

In stenosis of the duodenum or jejunum, owing to the stagnation and decomposition of albuminous substances, the products of which (indol and phenol) are absorbed and partly excreted by the urine, use may be made of the discovery of increased amounts of indican in the urine for diagnostic purposes. On the other hand, in stenosis of the large intestine the urinary test may be negative, since the albuminous elements of the intestinal contents are absorbed before they reach the stenosed portion of bowel, where stagnation and putrefaction can take place.

Examination *per rectum* with the finger or with the rectal tube, by means of liquid distention or gaseous inflation of the colon, may enable us to determine the seat of obstruction in certain cases. The detection of a deeply-seated incarcerated hernia (in the abdominal fossæ and pouches, diaphragm, or obturator foramen) is often made only *postmortem*.

**Nature of the Obstruction.**—This is even more difficult of discovery than the preceding. The following causes of obstruction with their differentiation may be referred to in attempting a diagnosis: *Strangulation* often affords a previous history of peritonitis or abdominal section or of recurrent attacks of abdominal pain, occurring mostly in young adults. Early fecaloid vomiting is common.

*Intussusception* usually gives a negative previous history. The suddenness of the attack, without appreciable cause, occurring in a child, and associated with colicky pain, tenesmus, and the presence of mucus and bloody stools, and of an elongated cylindric tumor in the right iliac or umbilical regions, however, render this condition easy of diagnosis in some instances. It is to be noted that absolute constipation and meteorism here are unusual. The intussusception may be felt in the rectum.

In *volvulus* it may be helpful to elicit a history of former constipation and flatulence, with evidences of atony of the bowel, in persons of advanced years, along with marked abdominal tympany, tenderness over a distended coil, which may perhaps be outlined (Wahl), a rigid abdomen, and sometimes dyspnea from great gaseous distention.

The history in cases of fecal obstruction is nearly always one of obstinate, habitual constipation, and occurs especially in females and neurotic subjects. The onset is gradual; pain is less acute; and tympany and fecal vomiting are less prominent and late in appearance. Fecal masses in the colon and rectum may be palpated, and even indented, particularly in the cecal and sigmoid flexures. Dulness is present on percussion, with slight tenderness over the tumor.

Obstruction due to *large enteroliths* or *foreign bodies* may be only surmised; especially is this true when symptoms of appendicitis arise.

*Biliary calculi* may give a history of previous attacks of hepatic colic and jaundice.

In the chronic obstructive form of *stricture* of the bowel due to cicatrices or neoplasmata the history of dysentery, tuberculosis, sarcoma, or carcinoma should be considered. The detection of an irregular tumor and the cancerous cachexia point to malignancy.

In obstruction caused by *intestinal paresis* there is generally a history of a previous enteritis, peritonitis, or celiotomy. The abdomen is smooth, though tympanitic throughout, and there is no perceptible peristalsis.

Not rarely it will be of therapeutic as well as of diagnostic importance to ascertain whether an attack of acute obstruction is primary, or whether it is the terminal exacerbation of a chronic condition, such as carcinoma of the bowel. Here a study of the past history of the patient, as well of the present signs of a probable nature, will afford considerable aid.

**Differential Diagnosis.**—Acute intestinal obstruction must be discriminated from *acute generalized peritonitis*.

#### ACUTE GENERALIZED PERITONITIS.

#### ACUTE INTESTINAL OBSTRUCTION.

##### *Etiology.*

There is a history of causal conditions or diseases (ulcer, appendicitis, pelvic infection).

There is a history of previous chronic obstruction or hernia. (The age of the patient if it be intussusception.)

##### *Symptoms.*

An early and considerable rise of temperature; later variable or may be absent. Pain more continuous and diffuse. Vomiting is characteristic, but not stercoraceous. Collapse occurs later. Slight increase of indican in the urine.

No early rise (except in volvulus), but later with advent of peritonitis. Pain in short paroxysms and localized. Vomiting becomes characteristically stercoraceous. Earlier onset of collapse. Excessive indicanuria, particularly when the small intestine is obstructed.

*Physical Signs.*

Distention of the abdomen is usually general and marked.	Less marked (sometimes partial), unless the obstruction be situated in the lower segment.
Visible peristaltic waves absent.	Present and pronounced when the seat of obstruction is low.
Tenderness general.	Tenderness localized.
Signs of effusion appear.	Less common, due to secondary peritonitis.
Auscultation negative.	Loud gurgling and splashing sounds audible on auscultation.
Prognosis almost hopeless.	Not so if operated upon early.

It must also be differentiated from *acute enteritis*, in which (particularly when due to toxic minerals) there is more apt to be diarrhea with considerable mucus and blood, an elevated temperature, intense gastric pain, associated with traces of the poison in the vomitus, as well as with its effects on the oral mucous membrane, and an absence of marked tympanites and fecal vomiting. There are also localized pain, tenderness, and tumor, or there may be collapse.

The various forms of *abdominal colic*, as enteralgia, hepatalgia, and nephralgia, should not be mistaken for acute intestinal obstruction after considering the history of the cases, the character and locality of the pain, and the absence of such symptoms as obstinate constipation, fecal vomiting, early collapse, intense local pain and tenderness.

**Course, Complications, and Prognosis.**—A case of acute obstruction usually terminates within from two to seven days. The chronic form may last weeks, and even months, with progressive emaciation and anemia, until the superaddition of more or less acute symptoms, lasting from ten to fourteen days. As a rule, the *prognosis* is wholly unfavorable, and especially in the acute cases. The chronic forms, due to fecal or other impaction, often recover with the discharge of the disturbing intestinal contents. Life may be prolonged by surgical interference in certain cases if they are taken in their inception.

*Complications* that may occur, as secondary peritonitis, gangrene, perforation, septico-pyemia, and enteritis, are all grave, and only tend to hasten the dissolution.

**Treatment.**—Whilst the treatment of intestinal obstruction is sooner or later essentially surgical, attention to the medical aspect is frequently of prime importance. The first indications for therapeutic interference in acute obstruction are presented by the *pain* and the *incessant vomiting*. The former is to be met by hypodermic injections of morphin, which at the same time tend to arrest the excessive peristalsis. For the vomiting no other measures are comparable to gastric lavage and starvation. It is well in most cases to withhold food for some hours to prevent retching and aggravation of the condition. The lavage is strongly advised by Kussmaul, who claims that both the tension above the seat of stricture and the inordinate peristalsis are thus greatly diminished and, exceptionally, cured. It may be repeated every six hours. A diagnosis of intestinal obstruction having been made without having learned the cause or character of the obstruction, cathartics should absolutely not be given. If it has been determined that fecal impaction is the trouble, it is still prudent to avoid purgatives until the



main mass has been moved, as in many cases there are both paresis and inflammation at the seat of impaction, so that this class of agents would thus be useless, if not harmful. High rectal injections, copious, steady, and regularly repeated, are to be practised, using for this purpose preferably "a warm saline solution of olive oil" (particularly if scybala be present) administered while the patient is in an inverted position by means of a fountain syringe, so that the flow is readily controllable. The abdomen should be methodically kneaded (a valuable adjunct in the procedure) and the patient at times well shaken. This method of treatment, by hydrostatic pressure, can and must be carried forward without undue violence, and if it be unsuccessful, the intestines are to be inflated from a large india-rubber bag with air or hydrogen gas (Senn), of which two to three gallons may be cautiously introduced. Thorough manipulation of the abdomen from below upward, particularly if it be a case of intussusception, may be combined. In the latter condition inflation, early and perseveringly applied, cures the majority of instances. In cases of intussusception or strangulation of the bowels these efforts should be continued for twenty-four hours, when, if the condition is not relieved, immediate operation is to be encouraged and advised. Although the statistics of Fitz show the mortality in cases without operation to be lower (69 per cent.) than with operation (83 per cent.), I am convinced from personal observation that the less favorable results from abdominal section would not obtain if it were performed in due time. To relieve the excruciating tympanites the plunging of a fine trocar and cannula into the intensely distended bowel, as in case of volvulus, may be required.

In chronic obstruction the treatment of the underlying or etiologic conditions and various complications is to be conducted on general principles. Additionally, the patient's dietary is to be arranged with care, and the bowels moved with unfailing regularity, by the use of unirritating laxatives and enemata. During the periods of threatening complete occlusion, with pain, the methods advocated above for acute obstruction are appropriate. If total obstruction persist despite medical treatment, surgical treatment—enterectomy, enterotomy, or other operation, as the circumstances of individual cases may dictate—is required.

The after-treatment consists in keeping the bowels active and regular by habit, diet, and an aperient pill if needed. Massage and electricity to the abdomen are found useful at this time.

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## CARCINOMA OF THE INTESTINE.

(*Carcinoma Intestinalis*.)

CARCINOMA of the intestine is perhaps the commonest cause of chronic intestinal obstruction. The stenosis is usually partial, and is due both to compression and to direct invasion of the lumen of the bowel by the growth. Primary intestinal carcinoma is rare in comparison with the occurrence of gastric carcinoma.

**Pathology.**—When carcinoma attacks the intestine it is usually in the form of a cylindric-celled epithelioma, although it may assume the

various forms as found in carcinoma of the stomach—namely, scirrhus, medullary, and colloid. The growth may be annular or semipolypoid, or it may occur as a diffuse nodular infiltration of the bowel-walls. Ulceration of the surface of the carcinoma may take place, and the glandular structures of the abdominal cavity sometimes reveal metastatic growths. The most frequent seat of intestinal carcinoma is the rectum, and next in order of frequency are the sigmoid flexure, the transverse and descending colon, the *papilla duodenalis*, the ascending colon, and the lower and middle portions of the ileum. The bowel is dilated above the constriction, and is usually filled with an accumulation of fecal matter. The muscular coat is hypertrophied. Below the narrowing the intestine may be small and atrophied.

**Etiology.**—Hereditv and advanced age are of chief importance as predisposing causes. Whether or not antecedent intestinal ulceration may afford a probable nidus for carcinomatous growths is doubtful.

**Symptoms.**—A description of the course of *rectal carcinoma* belongs more properly to surgical works. The chief symptoms are *progressively increasing distress* and *radiating pain* in the *rectum*; these occur, at first, during defecation only, but later almost constantly. There may be diarrhea alternating with constipation, and the feces often contain blood and mucus. Gradual *bodily wasting* and increasing mental anxiety are associated. Paralysis of the anal sphincter and consequent incontinence may ensue.

The symptoms of carcinoma of the bowel *above the rectum* are often vague, and vary according to the portion involved by the neoplasm. With or without an appreciable tumor in the abdomen the clinical history is usually that of *chronic obstipation* of the intestines. There are irregular attacks of *sharp, colicky pains*, especially a few hours after eating, distressing defecation, obstinate constipation, perhaps alternating with diarrhea, sometimes vomiting, which may be feculent, and not rarely slight meteorism. The special symptoms of carcinoma of the *papilla of Vater* are vomiting, jaundice, and colic. The progressive emaciation and debility of the patient are marked. In advanced cases of stenosis the *feces* are passed in small, compressed lumps resembling sheep's dung.

**Physical Examination.**—*Inspection* of the abdomen may show the presence of a tumor produced by the carcinomatous growth along the line of the sigmoid flexure or colon; peristalsis may be seen above the site of the carcinoma, communicating its movements to the abdominal walls. *Palpation* may be resorted to in order to confirm the above, and the growth is then frequently found to be nodulated. *Percussion* may give either dulness or a muffled tympany over the tumor and for some distance above, on account of accumulated masses of feces. This area of impaired intestinal tympany may be sharply defined by a clear tympanic note elicited over the empty bowel below the growth.

**Diagnosis.**—This may rest, in some cases, upon heredity, the age, the evidences of the cancerous cachexia, sharp, radiating abdominal pains, bloody stools, and the detection of a more or less firm and nodular tumor.

**Differential Diagnosis.**—(a) Carcinoma of the bowel above the rectum needs to be discriminated from other abdominal tumors. The presence of the following may render the diagnosis of carcinoma during life well-nigh impossible: sarcomata, fibromata, myomata, adenomata, and cys-

tomata, all of which may produce symptoms of obstruction like those due to carcinomatous growths. The cancerous cachexia may be simulated by other conditions. The advanced age of the patient and the distressingly rapid and downward progress of the disease will, however, point toward malignancy. *Fecal tumors, enteroliths, and foreign bodies* may need to be excluded also. Fecal masses have been mistaken for carcinoma, and when it is recollected that such may exist above and overshadow the presence of carcinoma of the intestine, the difficulty in differentiating the two is quite obvious.

(b) *The portion of the bowel involved* by the neoplastic growth is also difficult of definite diagnosis, except when it occurs in the rectum, when the digital and visual examination of the parts, supplemented, it may be, by microscopy, are sufficient. The locality of the tumor as detected by palpation, associated with special symptoms, is of value in arriving at a diagnosis of the diseased portion of bowel. Heulin<sup>1</sup> has studied carefully primary cancer of the duodenum, and asserts that the comparative frequency of duodenal involvement is due to limited motion of the organ, being thus subject to injury. When it occurs above the papilla of Vater the symptoms greatly resemble those of *dilatation of the stomach*. An important point separating carcinoma above from that below the papilla is the presence or absence of bile in the vomit, being absent if situated above. When the carcinoma involves the papilla of Vater symptoms of biliary obstruction necessarily follow. A hard nodular mass may sometimes be felt in the lower epigastric region; this coupled with increasing gastric dilatation and marked persistent jaundice would indicate carcinoma of the duodenum. It is apparent, however, that *carcinoma of the pylorus*, of the left lobe of the liver, or of the omentum or mesenteric glands, or a thickened cecum might all be easily confounded with carcinoma of the bowel at various adjacent parts of its course. The injection of fluid into the bowel may be resorted to in locating the probable situation of the growth. Thus, if obstruction from carcinoma exists in the sigmoid flexure, liquid will be arrested there and the rectum distended; while, if the stenosis be high up in the large or small intestine, the colon will be found comparatively emptied of feces and will be distended with the injected liquid.

**Course and Complications.**—Carcinoma of the intestine sometimes runs a rapid course, and, symptomatically at least, lasts but a few months or even weeks; in the scirrhus variety, however, the disease may last two or three years.

Intestinal carcinoma may perforate the bowel and cause fatal purulent peritonitis, and carcinoma of the rectum may perforate and invade the vagina and bladder, causing purulent vaginitis and cystitis. Or, owing to extreme distention by fecal accumulation between a cancerous stricture of the sigmoid flexure, for instance, and the resistant ileo-cecal valve, rupture of the colon, followed by a terminal peritonitis, may result. Extension of the growth into surrounding tissues, with ulceration, may lead to cellulitis, phlebitis, and pyemia.

The **prognosis** is almost hopeless.

**Treatment.**—This, from a strictly medical standpoint, is simply

<sup>1</sup> *Gaz. hebdom. de Méd. et de Chir.*, Feb. 13, 1898; *Thèse de Paris*, 1897; *Saunders' Year-Book*, 1899, p. 194.



palliative. The diet should be highly nourishing and easily assimilable, but when the symptoms of acute obstruction supervene the administration of food by the mouth is contraindicated. Attention to the state of the bowels by the use of enemata, or of the aloin, strychnin, and belladonna pill is necessary in most cases. Opium or cannabis indica for the pain, and stimulants for the depression, may also be serviceable.

Lavage of the stomach gives decided relief when decomposing matters tend to cause regurgitation on account of the damming back of accumulated food-detritus.

Carcinoma of the bowel may be treated surgically by colotomy, excision, lateral anastomosis of the bowel, enterostomy, and, if the growth be situated in the rectum, by extirpation by means of sacral resection (*Kraske's operation*).

## HABITUAL CONSTIPATION.

(*Costiveness.*)

**Definition.**—Chronic fecal retention, habitual infrequency, irregularity, difficulty, or insufficiency of the evacuations of the bowels.

Although constipation is a symptom, and although habitual constipation is frequently a symptom of chronic disease, the causal elements of the latter may be so indefinite and obscure that the former takes on all the individual importance of a functional affection. I describe habitual constipation, therefore, as a disease *sui generis* ("idiopathic").

**Etiology.**—In the majority of cases habitual constipation is the direct effect of a lack of expulsive or peristaltic power, and also of a deficiency of the hepatic and intestinal secretions. Two sets of causes operate to bring about these conditions of abnormal defecation:

**General Causes.**—(a) *Temperament*: it has been observed often that people of a nervous and "bilious" or motive temperament, of the dark type—brunets with a predominating nervo-muscular susceptibility—are much troubled with inherent constipation. Anemic brunets—persons having pale skin and dark hair combined—are particularly so affected, although alternating periods of diarrhea may supervene, owing to the hydremic state of the blood. "Torpid liver" and "sluggish bowels" are commonly held to be synonymous with these physical characteristics. (b) *Habit*: a sedentary life conduces to secretive inactivity. Thus, a lazy life, in which the calls of nature are irregularly attended to or habitually neglected, leads to frequent over-distention of the rectum and paresis, a common cause of chronic constipation. Again, the feminine false modesty (so called) that prompts a postponement and suppression of the desire to defecate in public places, as well as the habitual, hurried performance of the act in illy-kept, uncomfortable, and unsanitary closets,—all these tend to obtund the sensibility of the bowel to fecal masses in the rectum. The accumulation of these fecal masses causes paralytic over-distention, their hardening into scybala, and difficulty of expulsion. (c) *General bodily weakness, and diseases*, as neurasthenia, hysteria, anemic brain- and spinal-cord affections (causing inhibitory disturbances of the intestinal nerve-supply), acute fevers, hepatic disorders, especially the presence of jaundice, and the habitual

dependence upon and use of purgatives. (*d*) *Diet*: the constant use of concentrated articles of food, as meats, in which little residual matter is left to stimulate the bowel to peristalsis. On the other hand, a very coarse diet may leave such an excess of residue as to cause fecal impaction. (*e*) *Abundant and prolonged diuresis and diaphoresis*, by causing loss of fluids, also may induce chronic constipation.

**Local Causes.**—(*a*) *Atony of the abdominal muscles* from obesity or, in females, as a result of many pregnancies. (*b*) *Atony of the large bowel* (the sigmoid flexure in particular) from chronic colitis. (*c*) *Pressure by tumors*. (*d*) The presence of *intestinal stenosis* from external or internal constriction. (*e*) *Congenital stricture or giant growth* of the colon, with coprostasis (as in Formad's case) (Functional Neuroses of the Intestines, *vide* p. 846).

**Symptoms.**—In cases in which there is no adequate cause for habitual constipation other than a constitutional and perhaps an inherent peculiarity there may be the appearance of perfect health. Nothing is complained of save the fact that an evacuation of the bowels occurs too infrequently. It should be borne in mind here, however, that the term "constipation" is, individually speaking, almost wholly a relative one—*i. e.* one person may enjoy good health with but one evacuation every other day, another with two passages per diem, while still another must have one stool a day, *ceteris paribus*, to feel perfectly well. The last is usually considered an average normal state with most people. Persons such as are instanced above, in apparently good health, but observing that they have to defecate less often than many others, sometimes grow anxious, worried, and even hypochondriac, until assured that they are not truly constipated if enjoying perfect physical ease.

Symptoms of habitual constipation may be direct or reflex. *Direct* or *local* troubles are seen in the feeling of fulness, weight, and pressure in the perineum and abdomen. Flatulence, colicky pains, and alternating diarrhea occur not infrequently. The hurried and inattentive performance of defecation gives rise to the so-called "cumulative constipation," in which the accumulated feces are but partially evacuated with the movement, and the rectum consequently is not emptied. A sense of fulness then remains, and complete relief is not felt in these cases.

*Reflex* and *general* symptoms are malaise, languor, hebetude, irritability of temper, headache, facial flushing, palpitation, cold extremities, anorexia, vertiginous attacks, paresthesia, menstrual distress in women, sleeplessness, and bad dreams. Pressure on the sacral and visceral nerves may cause neuralgias. The tongue is coated. Palpation of the abdomen often shows the presence of doughy-like fecal tumors at the cecum or at the hepatic, splenic, and sigmoid flexures, or of bologna-like masses at intervening places. In marked cases attacks of nausea and vomiting, with diarrhea, may ensue; fever may also be present, and typhoid fever even may be simulated (Meigs).

**Complications and Sequelæ.**—Hemorrhoids, ulcerative colitis, perforation, and enteritis may be associated with chronic constipation. Not rarely do we have as results dilatation of the colon or sacculations, with the presence, in old people mainly, of *enteroliths* (calcified scybala); also intestinal obstruction and typhlitis, or cerebral hemorrhage or hernia from violent straining efforts.

**Diagnosis.**—Bearing in mind the relativity of constipation in different individuals, the diagnosis is read at sight. The detection of the causes is not difficult, though sometimes tedious. Hypochondriasis or melancholia should be carefully placed either as precedent to or consequent upon chronic constipation, the nervous condition often acting to produce the latter, and *vice versâ*.

The **prognosis** is usually favorable, but should be guarded.

**Treatment.**—**Hygienic.**—Causative factors must, of course, be removed, modified, or lessened. Systematic regularity as to time and frequency and sufficiency of movements of the bowels should be enjoined upon and practised by the patient. Exercise is of signal value, and particularly horseback riding or gymnastic motions that bring the abdominal muscles into play. Attention to the calls of nature should be esteemed a duty, and proper time and heed must always be given to the completeness of defecation. Young girls especially should be instructed in this regard. The wholesale swallowing of cathartics is to be vigorously combated. The dietetic regimen, if properly looked after, often avails much in relieving this affection, and foods calculated to be easily digestible, but leaving a moderate residue after digestion, are to be recommended. Such are bread made of unbolted flour, plenty of vegetables and fruits, butter, and such laxative articles as figs or honey. A glass of cold water taken regularly at bed-time and in the morning before breakfast is efficacious and a point of common knowledge.

**Remedial.**—The methods and means offered for the cure of chronic constipation number legion. From the little aperient pill or “peristaltic persuader” to the cannon-ball rolled externally along the course of the large bowel is made up such a list of drugs and measures as to leave untenable any plea of lack of resource that may be advanced. *Drugs occupy a subordinate part in the treatment of habitual constipation.* Indeed, their use should be restricted mainly to those periods when the bowels become unusually obstinate and when a more or less free movement is urgently needed. That the constant use of laxative and purgative drugs tends to a confirmation of the condition, and its ultimate resistance to the action of cathartics when circumstances will have required their use, is familiarly known.

I have found of value in lithemic and dyspeptic subjects the laxative bitter waters (Hunyadi Janos, Kissingen, Friedrichshall, Carlsbad).

Drugs employed to unload a filled bowel may at times be used sparingly and in the smallest adequate quantities; the mildest forms should be selected. Since the constipation is only temporarily relieved by catharsis, the frequent use of strong purgatives in large doses only tends to render the bowel accustomed to their use. Creosote in large doses has recently been highly recommended; it probably relieves the constipation by overcoming the intoxication.

Among those laxatives and cathartics most commonly used may be mentioned aloes, rhubarb, Rochelle and Epsom salts, compound licorice powder, castor oil, jalap, senna, mercury, colocynth, and podophyllin. Important adjuncts in combination with one or more of the above are the extract of *nux vomica* (or strychnin) and the extracts of belladonna, hyoseyamus, and physostigma. The much-used aloes, strychnin, and belladonna pill can be used for a considerable length of time in the hope



of stimulating a normal intestinal and sphincteric activity, and thus inducing even a cure in some cases. The formula may be made up as follows :

R <sub>y</sub> . Aloin.,	gr. iij-v (0.194-0.324);
Strychninæ sulphat.,	gr. $\frac{1}{3}$ - $\frac{1}{2}$ (0.0216-0.0324);
Extr. belladonnæ,	gr. ij-ijss (0.129-0.162).

M. et div. in pil. No. xx.  
Sig. One pill at bedtime.

Sulphur in confection, along with the official pill of aloes and iron, has been recommended for the habitual constipation of anemia. In senile atony of the bowel, with much flatulence, a laxative pill having in combination asafetida or capsicum is often beneficial.

The subjoined formulæ are also rationally and empirically serviceable in chronic constipation :

R <sub>y</sub> . Ext. cascar. sagrad.,	ʒss (2.0);
Ext. nucis vomicæ,	gr. iv (0.259);
Ext. physostigmat.,	gr. iij (0.194);
Ext. belladonnæ,	gr. ij (0.129).

M. et ft. in pil. No. xx.  
Sig. One at night, or night and morning.

(Aloes, gr. j (0.0648), or podophyllin, gr. ij-ijj (0.129-0.194), may be substituted for cascara in the foregoing formula.)

Or,

R <sub>y</sub> . Ext. colocynth. comp.,	gr. xxx-xl (1.94-2.59);
Ext. hyoscyami,	gr. x-xx (0.648-1.29);
Ext. nucis vomicæ,	gr. iv (0.259);
Ext. gentianæ,	gr. xx (1.29).

M. et ft. in pil. No. xx.  
Sig. As above.

The **mechanical** means of relieving habitual constipation, as by *enemata*, are injurious if long continued, by reason of their irritating effect on the rectal and colonic mucous membrane, as well as on account of their tendency to become incompetent. At times, when the stomach is weak or irritable, a loaded bowel may be relieved by an ordinary enema of soap and water or by one containing  $\frac{1}{2}$  to 1 ounce (16.0-32.0) of castor oil, with 1 or 2 drams (4.0-8.0) of oil of turpentine if there be some flatulence. Glycerin enemata, containing from  $\frac{1}{2}$  to 2 ounces (16.0-64.0) of the agent, may be used. *Suppositories* of soap, molasses candy, or glycerin are included in the armamentarium. *Massage* also claims an important part in the relief of habitual constipation. It acts by stimulating the peristalsis and the abdominal muscles, and should be employed at set times in the day preceding a desired evacuation of the bowels. The hand of the *masseur*, or that of the trained patient even, when systematically used in this way, may be effectual when all other means have failed. The regular rolling of a metal ball along the course of the greater gut may be mentioned for its novelty as well as for its undoubted efficacy. The application of the faradic current to the abdominal walls or galvanization of the lumbo-abdominal

circuit deserves proper trial in many cases. Hydro-therapeutic measures, or cold sponging and baths, are nearly always useful adjuncts in the treatment of this often stubborn affection.

## DILATATION OF THE COLON.

(*Ectasia of the Colon.*)

THIS is usually a chronic condition, though not rarely it is acute. It may also be general, but in the majority of cases it is confined to the colon, and particularly to the sigmoid flexure. The *postmortem* findings are those of hypertrophic dilatation of the bowel, and rarely (as in a case of Rolleston and Hayward<sup>1</sup>) ulcerative and catarrhal lesions of the intestinal mucosa are noted. The sigmoid flexure is prone to become dilated in subjects in whom it is congenitally elongated. Mya<sup>2</sup> believes that the condition is due to a faulty development and not to fetal disease. The most distinctive features are *constipation*, which generally dates from infancy, and great *abdominal distention*. In the case of Rolleston and Hayward peristaltic waves were visible upon the surface. The condition may fluctuate, constipation alternating with regular daily movements, and the distention changing to a normal softness of the abdominal parietes in some instances. I have recently seen a case of this kind in a male aged twenty-seven, in whom the affection had commenced in infancy. In the *treatment* of the constipation resulting from congenital ectasia of the colon, lavage of the intestine with a very long tube is superior to laxatives or purgatives.

## NEUROSES OF THE INTESTINE.

As in the case of the stomach, these embrace derangements of (*a*) secretion, (*b*) sensation, and (*c*) motion.

### (*a*) SECRETORY DISTURBANCES.

Unquestionably the intestinal secretion may, through a purely nervous influence, be augmented. This manifests itself most frequently in the primary morbid secretion of mucus (*mucous colic*) and in membranous enteritis. Moreover, the fact that an actual catarrh of the intestinal mucosa may supervene as a secondary event is undeniable.

#### MEMBRANOUS ENTERITIS.

(*Enteritis Membranacea.*)

**Definition.**—A peculiar pathologic condition, chiefly of the large intestine, attended by a morbid secretion of mucus.

**Pathology.**—In the truly primary form there are no morbid lesions

<sup>1</sup> *British Medical Journal*, May 30, 1896.

<sup>2</sup> *Lo Sperimentale*, 1894, fasc. iii. p. 215.

discoverable in the mucosa. Osler states that the membrane is due to a derangement of the functions of the mucous glands the nature of which is unknown. My own view is that this is a secretory neurosis, and that the catarrhal process may develop as a secondary event.

**Etiology.**—Sex has a decided influence; according to W. A. Edwards, not less than 80 per cent. of all cases occurring in adults are noted in women. Hysterical females and those of a highly neurotic constitution are the most frequent victims of the disease, which is rare in children.

**Symptoms.**—I have found the condition to be invariably associated with a decidedly constipated habit—a fact that may, in part, explain its occurrence, since time is thus allowed for the formation of the membrane. The most important clinical feature is the *passage*, at *varying intervals*, of *long, ribbon-like threads of mucus*, or of more or less *perfect casts of the gut*, the act being attended with *tenesmus* and severe *colicky pains*. The composition of the stools has been thoroughly investigated by M. Rothmann and O. Rothmann and C. Ruge. They “consist of a uniformly turbid ground-substance, which, on the addition of acetic acid, becomes opaque and striped. It is interspersed with a cellular detritus, consisting partly of strongly refractile granules and partly of cellular elements, desquamated epithelial cells, round cells, and peculiar glossy flakes. There are also found cholesterin-crystals, needles of fatty acids, triple phosphates, remnants of undigested food, pigment-granules, many bacteria, and occasional red and white corpuscles.”

The individual paroxysms vary in *duration* from one to ten days or more. In one case observed by me the attacks lasted about two days, recurring regularly at the end of every three months. Ordinarily the recurrence is after a shorter interval.

**Diagnosis.**—It is important to make a microscopic examination of the pieces of membrane. If, when thus examined, mucus, cylindrical-celled epithelium, a few round cells, and the other elements already mentioned are found present, the diagnosis of mucous enteritis is undoubted. It is to be recollected, however, that membranes are not passed with every attack.

**Course and Prognosis.**—The disease pursues a very chronic course and lasts for many years. The bodily nutrition suffers considerably if the attacks are frequent and severe, though, as a rule, this does not occur until a late stage in the affection. The risk to life, it is needless to say, is slight.

#### (b) SENSORY DISTURBANCES.

It may be noted here that the sensory nerves of the intestines, as well as the inhibitory and vaso-motor dilators, are traceable to the splanchnics. Increased sensibility of the sensory nerves produces—

#### ENTERALGIA.

(*Neuralgia of the Intestine.*)

**Etiology.**—This is commonly met with in hysterical, neurasthenic, and anemic subjects. It occurs as a reflex neurosis, as in the case of



cold, gout, and irritative lesions of the pelvic organs (kidneys, liver). Enteralgia is symptomatic of many local affections and conditions that induce direct irritation of the sensory nerve-filaments of the intestine; among these are inflammation of the mucosa, foreign bodies, gall-stones, abnormal distention with gas, and enteroliths. Under these circumstances the condition is associated with increased activity of the motor nerves or heightened contraction of the muscularis, forming true intestinal colic. In lead colic it is probable that the lead acts directly upon the nerves or their ganglionic cells. I have repeatedly observed the action of certain exciting causes, and particularly of nervous shocks.

**Symptoms.**—Enteralgia may develop very *suddenly*, but oftener it sets in less abruptly, and is then attended with eructations of gas, expulsion of flatus, and the like. In the fully-developed, attack the *pain* may attain to great violence, causing the patient to “bend double” or even faint, and its character is variously described as boring, tearing, or cutting. The pain may be confined to a circumscribed spot or may be diffuse. The attacks are sometimes brief, or they may be characterized by a sudden subsidence. At other times they last for days or perhaps weeks, and then subside gradually. *Recurrences* are common, but the intervals between the attacks vary extremely in duration.

*Hypogastric neuralgia* is a term applied to neuralgia affecting the sensory nerves lying in the most dependent segments of the intestine. Here the nerve-fibers entering into the hemorrhoidal plexus are involved. It is caused chiefly by tabes, by hemorrhoids, and by the neurotic state so common to females. This form of neuralgia has its seat in the hypogastric region, and is accompanied by a distressing sensation of pressure in the rectum and bladder, and by an irresistible desire to go to stool; pains also radiate to the sacrum, thighs, and perineum.

**Diagnosis.**—The various organic diseases and conditions mentioned under Etiology, in the course of which colic is a common symptom, must be separated from the true neurotic enteralgia. The former are distinguished from the latter by a group of symptoms peculiar to themselves (fever, aggravation of the pain upon pressure, vomiting, constipation, or diarrhea), and by the usual definite causes furnished by the history.

*Renal and hepatic colic* bear a superficial similarity to enteralgia. The former conditions, however, are distinguished first by the seat and direction of the pain, and secondly by the appearance of jaundice in hepatic colic and of hematuria in renal colic. *Rheumatism* of the abdominal muscles is easily eliminated, since it is generally combined with rheumatism in other parts of the body; the pain is also greatly increased upon throwing the muscles into contraction, as in stooping or rising; finally, it vanishes in response to the action of the salicylates.

#### DIMINISHED INTESTINAL SENSIBILITY.

This implies diminished peristalsis or constipation. A greater or less degree of anesthesia of the bowel attends, with a loss of desire to go to stool and an accumulation of feces in the rectum. This is a usual concomitant in many diseases of the brain and cord, with which paralysis is associated. Motor innervation may remain intact, and when atony

of the intestine is absent spontaneous movements of the bowels occur; when atony is present, however, to a marked degree (motor paralysis), the feces must be artificially removed.

### (c) DISTURBANCES OF MOTILITY.

When the contractility of the muscularis is increased from purely nervous causes the result is—

#### NERVOUS DIARRHEA.

This condition presents no morbid lesions. The increased contractility results from an exaggerated irritability of the motor nerves of the bowels. It may also result from morbid processes in the central nervous system and in other organs of the body; in short, the condition may be a reflex one.

Examples of this sort are caused by tabes, by gastric disturbances, as after certain foods and drinks, by dentition, and the like. Most cases, however, are encountered in persons having an abnormally irritable nervous organization—*i. e.* the neurasthenic and hysteric classes. In such the effect of mental excitement, of fright, and similar psychic influences is to induce diarrheal evacuations.

**Symptoms.**—The *stools* vary in number from two or three to twenty-four or more daily. In rare instances they are soft—not truly diarrheal—and formed, yet they may be quite frequent. Blood and mucus, pus, and other morphologic elements are absent from the dejections. It is characteristic of nervous diarrhea that the stools follow one another in rapid succession, usually during the morning hours, and then discontinue for the greater part of the day. The bodily nutrition is often well preserved.

In the **diagnosis** organic affections of the bowel are to be carefully eliminated.

#### ENTEROSPASM.

(*Spasm of the Intestine.*)

By this term is meant a concurrent spasm of both the longitudinal and circular muscular fibers, usually inducing spasmodic constipation, and sometimes total, though temporary, occlusion of the bowel.

Its *causes* are similar to those of nervous diarrhea, and the condition is clinically related to enteralgia. Neither pain nor constipation, however, is a constant feature. The stools may assume the form of a ribbon or of large rounded masses (sheep's dung), but they are not pathognomonic. They may also be covered with mucus. Ewald distinguishes between an idiopathic and a secondary or symptomatic spasm, the latter being a concomitant of basilar meningitis and of chronic lead-poisoning. Another variety affects the rectum (*proctospasm*), and is generally secondary to some other rectal affection, as fissure of the anus; it may, however, occur as a neurosis in the hysteric and nervous class of subjects.

The *diagnosis* of true functional enterospasm can only be made after all organic causes that may produce spasm of the bowel have been excluded.

## CONSTIPATION.

This is a common condition as a functional neurosis. It is due to an abnormity of function of the intestinal nerves that leads to a weakened peristaltic action, and is met with in hysteria, neurasthenia, and in those suffering from the various forms of psychoses. Central nervous affections often manifest atony of the intestine as a symptom; hence this form is not a disease *sui generis*. Cases of this class do not respond to any variety of cathartics, whether they act upon the small or large intestine (Ewald).

Paralysis of the external sphincters is a common concomitant in a great variety of local (catarrhal) and central nervous diseases. Under these circumstances the act of defecation may be purely *reflex*, owing to loss of control of the voluntary muscles; or it may be *voluntary*, except when the person affected is not upon his guard, or during mental excitement, micturition, sneezing, and like influences, the latter condition being a mere weakness.

**Treatment of Intestinal Neuroses.**—A suitable change of environment, including an appropriate arrangement of the dietary, is of primary importance, and is uniformly applicable in this class of sufferers. Further, the treatment of special cases has peculiar reference to the character of the nervous derangement. After making an accurate diagnosis a search for the factors of the greatest etiologic importance should be made, and these must then be vigorously assailed.

In the *secretory neuroses* an associated membranous enteritis must be corrected, the digestion must be improved if faulty, and the obstinate constipation overcome. For the latter symptom enemata containing ox-gall, either alone or in combination with salines, are especially serviceable. Kussmaul and Fleiner have obtained the best results from regular large oil-enemata administered once or twice daily. During the painful attacks simple enemata, repeated every couple of hours, will sometimes bring speedy relief by facilitating the removal of the scybala, and will assist nature's efforts at separating the adherent membranes. Pain must be relieved by morphin.

In the *sensory disturbances* in which the activity of the sensory nerves is increased (enteralgia and hypogastric neuralgia) the treatment may be considered under two headings: first, the relief of the neuralgic pains; and secondly, the correction of the causes or conditions on which the enteralgia depends. If the pain be severe, opium or morphin may be required. Especially good as an antispasmodic is codein, which may suffice in all save the severer cases. The object should be to give the minimum amount of the opiate that will meet the necessities of the case, with a view to obviating a resultant constipation. In hypogastric neuralgia I have found suppositories containing opium to be little short of magical in their effects.

In cases in which there is constipation due to diminished sensibility, with a loss of motor innervation (atony of the bowel), the feces must be artificially removed unless the underlying condition can be successfully overcome. It is especially important that the environment—physical and psychic—be so regulated as to bring about an improvement in the gen-



eral condition of the patient. It may become necessary to employ tonic preparations of strychnin, iron, or arsenic.

The treatment of nervous diarrhea involves the same principles, so far as the indication presented by the peculiar nervous organization is concerned, as in the sensory and secretory neuroses. It is especially important to prevent the operation of the direct causes—fright, mental excitement. Astringents and intestinal antiseptics are not called for, unless the bodily nutrition be affected thereby. Enterospasm is to be met by the same remedies that are used to control enteralgia.

## IX. DISEASES OF THE LIVER.

### ANOMALIES IN SHAPE AND POSITION.

**Altered Shape.**—Occasionally **malformations** of the liver are met with that materially alter the shape of the organ, either primarily when the result of disease, or secondarily from pressure of adjacent structures. Of the latter class the most important cause is tight-lacing, met with almost exclusively in women and producing the so-called “corset liver.” The lower part of the right lobe of the liver is usually the part affected; the hepatic parenchyma is atrophied, owing to continued compression, and shows deep grooves that correspond to the position of the lower ribs. The connective-tissue capsule and the peritoneal coat are both thickened at this point, the smaller blood-vessels often being entirely obliterated. In marked cases the right lower lobe may become converted into a dense fibrous band, with only a vestige of the former liver-structure remaining. Among other acquired causes of anomalies in the shape of the liver may be mentioned deformities of the vertebræ and ribs, or tumors of the ribs or adjacent structures (the pylorus, omentum) pressing against the liver.

**Diagnosis.**—Rarely, clinical symptoms are present. “A constant *sensation of pressure and pulling* is felt in the hepatic region, and sometimes, as a result of venous stasis, there is a temporary but decided swelling of the isolated portion, and, possibly, *violent pain* and indications of irritation of the peritoneum, such as *vomiting* and an approach to *collapse*. Jaundice is rare in consequence of this deformity” (Strümpell). The danger of this condition lies in a possible mistaking it for an *abdominal tumor* (Pepper), *amyloid disease*, *passive congestion*, or *new growths* of the organ (Strümpell).

**Primary alterations** in the shape of the organ may be due to active or passive congestion, hereditary syphilis, hypertrophic or atrophic cirrhosis, acute yellow atrophy, carcinoma, abscess, or hydatid cyst. The accompanying *symptoms* would, of course, be those of the disease causing the deformity.

**Anomalies of position** are not infrequently met with, the organ being displaced upward, downward, or laterally. The most common cause of lateral displacement is found in an abnormal lengthening of the suspensory ligament. The organ may occupy the epigastric region or be dis-

placed into the lower part of the abdominal cavity, but a change in the posture of the patient or external pressure is often sufficient to replace the liver in its normal position. The *symptoms* (if present at all) consist of a dragging sensation, often amounting to pain that may be severe and referred to the right shoulder. On *physical examination* palpation may reveal a fissure between the right and left lobes, together with a movable tumor presenting the size and normal outlines of the liver, which by manipulation may be returned to the right hypochondriac region. Percussion gives tympany over the normal area of liver-dulness, which changes to flatness when the organ is pressed or falls into its natural position.

Displacement upward may result from gastric or intestinal distention, marked ascites, or an abdominal tumor; while downward displacement may be due to a mediastinal tumor, an emphysematous lung, or a pleural effusion.

**Diagnosis.**—Among the conditions likely to be confounded with movable liver may be mentioned *carcinoma of the omentum* or of the *pylorus*, *dermoid cysts*, *tumors of the ovary and uterus*, *hydro- or pyonephrosis*, *tumors of the kidney*, and *chronic proliferative peritonitis*. By a careful study of the symptomatology, and in the absence of the normal physical signs over the hepatic area, the differential diagnosis can usually be firmly established, although marked fatty degeneration or atrophic cirrhosis may coexist with any of the above conditions and cause marked diminution in the area of hepatic dulness.

The **treatment** of movable liver is merely palliative, and consists in the application of a suitable bandage for preventing the displacement.

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## JAUNDICE.

(*Icterus.*)

**Definition.**—A condition in which the tissues and secretions are stained with bile-pigments. Jaundice is not a disease, but a symptom. The doctrine of a hematogenous jaundice has been successfully overthrown by the investigations of Stadelmann, Hunter, and others. All forms are due to obstruction (hepatogenous); at all events the cases of hematogenous origin must be extremely exceptional.

*Hepatogenous or obstructive jaundice* is more commonly seen in—(1) Inflammatory swelling of the duodenum or of the lining membrane of the duct, which is by far the most common factor in its causation, and demands separate consideration (*vide infra*, Catarrhal Jaundice); (2) Foreign bodies within the ducts, as gall-stones or parasites; (3) Stricture or obliteration of the duct; (4) Tumors within the duct or obstructing its orifice; (5) Pressure on the duct from without, as by a tumor of the liver, stomach, pancreas, or omentum; also by fecal accumulations, displaced organs, a pregnant uterus, enlarged glands in the fissure of the liver, and, more rarely, by abdominal aneurysm; (6) Lowered blood-pressure in the vessels of the liver favoring resorption of bile, as in simple icterus of the new-born (Frerichs).

## CATARRHAL JAUNDICE.

(*Hepatogenous Jaundice; Icterus Catarrhalis; Duodeno-cholangitis; Inflammation of the Common Bile-duct.*)

**Definition.**—A condition characterized by a discoloration of the tissues from retention and absorption of bile and resulting from a catarrhal inflammation of the lining membrane of the ducts, more especially the larger, and of the duodenum.

**Pathology.**—On examining a liver and gall-bladder *in situ* the former is usually found enlarged, lighter in color than normally, and of a distinct icteroid tint. On making a longitudinal section drops of bile can be collected on the edge of the section-knife.

The gall-bladder is found distended with bile, and on firm pressure a tough plug of mucus is usually expelled from the common duct into the duodenum, after which bile flows into the intestine freely. The mucosa lining the *ductus communis* is swollen and inflamed, and the catarrhal process may extend to the cystic, and in some cases to the hepatic, duct. As a rule, that portion of the common duct lying in the intestinal wall is more frequently and more deeply involved. If the disease becomes chronic, a formation of connective tissue occurs, owing to the irritation caused by the retained secretion, and atrophy of the liver-cells, with biliary cirrhosis, may result. Suppuration is rare.

Toxic (hematogenous) jaundice, so-called, has for its lesion extensive catarrh of the intra-hepatic bile-ducts from their origin. Here duodenal catarrh is not necessary for the production of jaundice. It was formerly assumed that the pigment (hemoglobin) was liberated in the blood; but Stadelmann and others have shown that the bile containing the poison, or its irritant products (toxins), excite inflammation of the finer ducts.

**Etiology.**—As simple catarrhal jaundice results in a majority of cases from extension of inflammation due to gastro-duodenal catarrh, the principal *predisposing causes* are as follows: (*a*) Exposure to cold and wet; (*b*) The use of improper foods, under which heading may also be comprised faulty cooking and improper mastication; (*c*) The excessive or prolonged use of such irritants as tea, coffee, or alcohol; (*d*) Prolonged anxiety and mental or physical overwork; (*e*) Certain acute diseases, as pneumonia, relapsing fever, typhoid fever, and malaria (toxic jaundice, *vide supra*); (*f*) Portal obstruction, occurring in chronic heart- or kidney-disease; (*g*) More rarely it has occurred in epidemic form.

**Symptoms.**—Preceding the development of the distinctive features by several days, dyspeptic symptoms are in evidence (*vide* Gastro-hepatic Symptoms). The principal symptoms in detail are: (*a*) *Icterus*, or tinting of the body surface may be the first symptom noticed in this condition, appearing usually on the forehead and neck and rapidly spreading over the entire body. The conjunctivæ also early become discolored, and the general hue, though variable, is commonly a bright lemon-yellow. In chronic cases the color is apt to change to a bronzed or deep-green tint.

(*b*) *Secretions and Excretions.*—The urine and sweat are often found to contain bile-pigment, the patient's linen frequently being discolored. In extreme cases the urine may be dark-green in color, while in those of average severity it is of a lighter or deeper greenish-yellow hue. The shaken specimen foams, and the froth has a yellow color-tint.



Often the presence of bile is detected before any noticeable coloring of the conjunctivæ occurs. In cases of intense or long-standing jaundice albumin and tube-casts may be present, and the latter may be bile-stained.<sup>1</sup> Hyaline casts are often found in cases of moderate intensity.

The bowels are constipated, and the stools are pale-drab or slate-colored; they are usually very fetid. Diarrhea, however, may be present, owing to the production of irritating substances and decomposition.

The tears, saliva, and milk are rarely stained with bile-pigment. The expectoration also is rarely tinted, unless pneumonia or some form of pulmonary infiltration coexists.

(c) *Circulation*.—The pulse, although not appreciably altered in volume or tension, is usually slow (often 30 or even 20 beats per minute), though this is not an unfavorable symptom.

(d) The *temperature* is usually normal, although slight elevations may occur (100°–101° F.—37.7°–38.3° C.).

(e) *Gastro-hepatic Symptoms*.—Dyspeptic symptoms—viz. anorexia, a sense of fulness after eating with flatulence, acid eructations, nausea and vomiting, accompanied by a dull, heavy pain over the hepatic area, with some tenderness on pressure—are present. These often develop insidiously; more rarely they occur suddenly with a severe rigor or chill, violent headache, and vomiting—*e. g.* at the onset in the epidemic form.

(f) *Cutaneous Phenomena*.—Pruritus or itching often becomes a troublesome symptom, being more common, however, in the chronic forms. Lichen, urticaria, furuncles, and sweatings (diffused and localized) may develop, the latter being often limited to the skin covering the abdomen and the palms of the hands.

A peculiar disease of the skin called *xanthelasma* or *bita higoidea* may also occur. It consists of bright-yellow spots, slightly elevated, appearing on the eyelids, and rarely on other parts of the body.

In the more severe forms spots of ecchymosis, and in some instances profuse hemorrhages, may occur into the skin and mucous membranes. These are usually associated with other symptoms of a grave type.

(g) *Nervous Symptoms*.—Headache and vertigo are common; irritability of temper, despondency, and wakefulness or mental dulness almost equally so. With the oncoming of darkness vision may grow indistinct (*hemeralopia*) or it may attain unnatural clearness (*nyctalopia*). Rarely, objects look yellow (*xanthopsia*). The nervous phenomena observed in catarrhal jaundice are attributable to the effects of the bile-acids. In certain cases, however, associated with destruction of the hepatic substance, as in acute yellow atrophy, carcinoma, cirrhosis, and fatty degeneration, grave cerebral symptoms (acute delirium, convulsions, and coma) may develop suddenly and prove fatal. This class of symptoms has been named *acholia*, *cholemia*, or *cholesteremia* (the latter owing to the mistaken supposition that cholesterolin is the poisonous product). The true nature of the toxic agent

<sup>1</sup> *Tests for Bile*.—*Gmelin's test*, or the play of colors, consists in bringing a few drops of urine in contact with the same quantity of commercial nitric acid on a plain white slab, whereupon various shades of yellow, green, red, and violet are produced.

*Rosenbach's test* is made by filtering the suspected urine and touching the filter-paper with a drop of nitric acid. If bile be present, a green circle will form at the point of contact. (See also Choluria, p. 948.)

in the blood is unknown. In some fatal terminations of this character death was due directly to a renal complication.

The **physical signs** in a case of simple catarrhal jaundice show on palpation and percussion an increase in the hepatic area, the lower border of the liver projecting in some instances several fingers' breadths below the ribs. Rarely, the distended gall-bladder projects below the lower lobe of the liver, as when there is complete obstruction near or at the duodenum, and then it can be distinctly palpated.

**Diagnosis.**—The etiology (errors in hygiene and diet), a history of previously existing gastro-intestinal catarrh, the age of the patient (young adult life), and the appearance of the jaundice unaccompanied by pain or general emaciation, together with an absence of symptoms pointing to *cirrhosis*, *carcinoma*, or *acute yellow atrophy*, form a characteristic grouping of clinical indications.

**Duration and Prognosis.**—The *duration* of catarrhal jaundice varies from two to eight weeks. If the symptoms continue longer than two months, grave doubts may be entertained as to the case being one of simple jaundice. The prognosis is guardedly favorable. A rise of temperature usually indicates mischief (Pepper), while hemorrhages of the skin and mucous membranes always influence the issue unfavorably.

**Treatment.**—The *diet* and *hygiene* are the first considerations in the treatment. Rich, highly seasoned foods, rich pastries, fats, and sweets, are to be interdicted; starchy foods, lean meats, bread, soups (containing no fat), and green vegetables may, however, be used in moderation. Skimmed milk, butter-milk, and alkaline drinks (Vichy and Saratoga mineral waters) may be used freely, while sour wines, lemonades, and tamarind-water are allowable. Systematic bathing (Turkish or Russian baths, under supervision), regulated hours of sleep, and moderate exercise in the open air, all exert a beneficial effect. The free use of pure water often does good by increasing the flow of bile and by dislodging plugs of mucus that may obstruct the duodenum and the common duct.

Gerhardt and Kraus have recommended the faradic current, applied over the region of the gall-bladder; manipulation has also been tried with a view to removing the obstruction in the common duct. Neither of these methods has met with success.

The first *therapeutic indication* is to keep the bowels freely soluble by the use of saline aperients, as Hunyadi water or Carlsbad salts ( $\frac{1}{2}$  to 1 teaspoonful in hot water before meals). The latter remedies tend to lessen the catarrhal inflammation by depleting the mucous membranes. In obstinate constipation calomel, rhubarb, the extract of colocynth, or castor oil may be employed. Prevost and Binet believe that calomel is in part converted in the economy into mercuric chlorid, which stimulates the biliary secretion.

Conspicuous among other remedies may be mentioned the alkalies, sodium bicarbonate, salicylate, and phosphate, which tend to increase the flow of bile and render it less thick; hydrochloric acid (which, according to Ewald, by aiding digestion prevents the formation and consequent absorption of toxic substances), in combination with the bitter tonics—gentian, quassia, and nux vomica; ammonium chlorid, which sometimes proves beneficial; and silver nitrate (gr.  $\frac{1}{8}$ — $\frac{1}{4}$ —0.008–0.016, three times daily).

Injections of cold water ( $60^{\circ}$ – $70^{\circ}$  F.— $15.5^{\circ}$ – $21.1^{\circ}$  C.), daily, in quantities of 1 or 2 quarts (1–2 liters), are highly recommended as promoting the secretion of bile; while lavage, practised daily and over a protracted period of time (one to two months), has proved highly beneficial, especially when gastro-duodenal catarrh has existed. This treatment was advocated by Krull, but has given negative results in the hands of Osler and Burney Yeo.

*Itching*.—This troublesome symptom may often be relieved by the external application of a solution of borax or sodium bicarbonate ( $\bar{3}$ ss—Oj—16.0–512.0), or of menthol and alcohol (gr. x— $\bar{3}$ j—0.648–32.0). Internally, large doses of the bromids (gr. xx–xxx—1.29–1.94, at bedtime) or the continued use of pilocarpin (gr.  $\frac{1}{12}$  to  $\frac{1}{8}$ —0.005 to 0.008, two or three times a day), as recommended by Witkowski, are worthy of a trial.

*Flatulence*.—To this end it is important to regulate the diet, avoiding starches and sugars. Ox-gall and sodium chlorate (gr. v—0.324—of the latter three times a day). Charcoal tablets, bismuth subnitrate or salicylate, and beta-naphthol are all useful in checking fermentation. Irrigation of the colon with some efficient antiseptic in solution is often a factor of service.

*Diarrhea*.—Occasionally attacks of diarrhea alternate with constipation in catarrhal jaundice, and when present demand treatment. As they are usually due to fermentation, salol and creasote ( $\mathfrak{m}$ ss—0.033), combined with the bismuth salts (subgallate, subnitrate, subcarbonate, or salicylate), are usually efficacious; they are administered before meals.

*Headache* is caused by the circulation in the blood of some toxic principle, due to the absorption of bile; it is often persistent and annoying, although rarely acute in character. Temporary relief may sometimes be obtained as the result of free sweating induced by means of the hot bath or hot pack. Of drugs, caffein citrate, camphor monobromate, and phenacetin, either singly or in combination, may be recommended.

In the other forms of hepatogenous jaundice permanent relief can only be afforded by removing the obstruction in the biliary channels, and thus permitting the normal outflow of bile.

When the obstruction is due to mechanical causes (biliary calculi, tumors pressing on the duct) the treatment is chiefly surgical, and consists in their removal (*vide* Cholelithiasis).

#### OTHER FORMS OF JAUNDICE.

Modern experiments, as I have said (*vide* p. 848), tend to show that the so-called hematogenous jaundice is always hepatogenous—*i. e.* the blood-dyscrasia probably exerts a toxic influence on the liver-cells and intra-hepatic gall-ducts; and there may at the same time be a more rapid blood-destruction in the liver (Neumeyer, Stadelmann, *et al.*).

There are instances of jaundice in which active hemolysis is an element of importance (Stengel): (*a*) Severe infections, as septicopyemia, yellow fever, acute yellow atrophy of the liver, and the jaundice of the new-born. (*b*) Grave forms of anemia, as pernicious anemia and chlorosis. (*c*) Certain poisons, as the venom of snakes, chloroform- and ether-poisoning; also in poisoning by phosphorus, arsenic, mercury, and other minerals.



Experiments conducted by Münzer, Starling, Hopkins, and others, tend to show that the liver-function is not suppressed by many of the conditions and affections mentioned above; but, on the other hand, that increased secretion (*polycholia*) and increased formation of bile-pigments (*polychromia*) may prevail. Again, the poisons or toxins may cause swelling of the cells and compression of the biliary capillaries; this would cause obstruction of the outflow of bile and the subsequent absorption. Lastly, circulatory disturbances (emotional jaundice) may lead to overproduction, or obstructive retention, of bile.

A consideration of the **diagnosis, prognosis, and treatment** of this variety of jaundice is embraced in the description of the various causative disorders.

## BILIARY CALCULI.

(*Gall-stones; Cholelithiasis.*)

**Definition.**—Concretions formed in the gall-bladder, due to an altered physiologic function or pathologic change; they vary in their composition and consist for the most part of bile-elements, and often set up characteristic disturbances (*cholelithiasis*).

**Etiology.**—As a result of biliary retention increased consistency and a concentration of bile occurs, and certain constituents that were before held in solution are thrown down. Among the most common **predisposing causes** may be mentioned the following: (*a*) *Female sex*, especially between the ages of forty and sixty. Senac's statistics, out of a total of 311 individuals, give 227 women (Dujardin-Beaumetz). (*b*) *Irregular meals* and an *excessive diet of starches and of fats*, combined with a sedentary life, are strong predisposing factors. (*c*) According to Harley, gall-stones and biliary concretions of all kinds are frequently *hereditary*. Among other, and perhaps minor, causes are *constipation, tight-lacing, pregnancy, chronic obstruction* to the flow of bile (as from tumors, catarrh of the ducts, or heart-disease, as mitral stenosis), and, more rarely, the rachitic and lithic-acid diathesis. (*d*) It may occur during childhood. (*e*) **Incidence.** Brockbank found among 13,047 completed post-mortem records, 7.4 per cent. were gall-stones.

**Composition and Appearance.**—Water comprises from 2 to 5 per cent. of the composition of gall-stones, the chief solid constituent being cholesterin, and the remainder being composed of bile-pigment and salts (lime, potash, soda, and perhaps traces of iron and copper). *Pigment-lime* may be, though rarely, the main constituent. In *size* they vary from the smallest particle of sand to that of a goose-egg. Harley records a case in which a pyriform cholesterin-calculus was discovered in the feces; in a dry state it weighed 400 grains (26.0) and measured  $2\frac{1}{4}$  inches (5.6 cm.) in length and  $1\frac{1}{10}$  inches (2.7 cm.) in diameter. Fagge reports a calculus weighing, in a dry state, 462 grains (30.0). The *color* varies from white or light-yellow to that of a dark-green (as in pigment-lime calculi), and may present any variation

between these two extremes. The *nucleus* often consists of cholesterin, the outer layer being usually the harder, and made up, for the most part, of lime-salts. The center of the nucleus generally consists of desquamated epithelium or dried mucus, and on cross-section concentric laminæ are usually developed. The cholesterin gall-stones cut like wax, are white, and the cut section presents a crystalline appearance. Other forms are apt to be brittle. The *surfaces* may be smooth, striated, or hollowed out, solitary calculi being usually round or ovoid, while multiple stones often present smooth facets, due to the massing together of the calculi (Dujardin-Beaumetz). They are usually olive-shaped, but may be pyramidal, cylindric, lenticular, pisiform, cubic, finger-shaped, or olivary. Their *seat* is usually the gall-bladder, but they may be found anywhere along the biliary passages.

**Symptoms.**—There may be no subjective symptoms of biliary calculi unless the stone becomes impacted in the hepatic, cystic, or common duct. Thus, Naunyn states that “the gall-bladder will tolerate large numbers for an indefinite period of time, postmortem examinations showing that they are present in 25 per cent. of all women over sixty years of age;” and I quite agree with him in his estimate. The passage of a calculus through the duct will give rise to *hepatic colic*, whereas a permanent blocking of the duct will cause symptoms of chronic obstruction, followed in many cases by those of ulceration and perforation, with the establishment of a biliary fistula.

**Hepatic Colic.**—When a gall-stone becomes impacted in a bile-duct the patient experiences *agonizing pain* (tearing, cutting, or lancinating in character) in the right hypochondriac region, radiating to the right shoulder, and accompanied often by profuse *sweating*, *vomiting*, and a *feeble, running pulse*. The most common *seat* of the pain is two to three inches to the right of the median line and about an equal distance below the ensiform cartilage. Less frequently it is in the region of the gall-bladder. This happens in cases in which the gall-stone is impacted in the cystic duct, and may be due to distention of the gall-bladder, or to associated cholecystitis. The pain is sometimes so severe as to produce *syncope*. Hepatic colic, however, may occur independently of the passage of biliary calculi, as from inflammation of the gall-bladder. On the other hand, large calculi have been found in the dejecta without having excited hepatic colic. I recently saw an instance of this kind in which the gall-stone was of the size of an English walnut. A *rigor* or *chill* often precedes the attack, which is usually accompanied by moderate *fever*, the temperature reaching 101°–102° F. (38.3°–38.8° C.). If the stone passes through the duct without becoming impacted, jaundice and pain may either be only slight or entirely absent. When, however, occlusion of the common duct occurs, the *jaundice* becomes intense. This symptom, however, may be present, though less marked, before the gall-stones reach the *ductus communis*. Jaundice occurs in about 50 per cent. of the cases (Fitz), and it sets in from eight to twenty-four hours after the onset of the attack of pain. *Physical examination* reveals on inspection a slight prominence in the hepatic area, and on palpation the edge of the liver can often be distinctly felt below the costal margin—at times as low as the umbilical level. The enlarged liver is sensitive on pressure, and particularly the gall-bladder, which can be often pal-

pated. If the latter viscus contains many calculi, crepitation may be noticeable to the palpating fingers (rarely), and a friction-sound may be distinguished on auscultation. The swollen organ, after the cessation of the colic, quickly subsides. *Recurrences* of the painful attacks after varying intervals of time are common. Finally, the gall-stone is expelled and the colic ceases to return. Multiple stones, however, may be passed.

*Rupture of the duct*, followed by fatal peritonitis, has been known to occur. Attacks of biliary colic are of variable *duration*, lasting from a few hours to a few days, and in some instances one or more weeks. Sudden cessation of the pain is usually followed by rapid disappearance of the jaundice (when present) and the discovery of the *stone in the feces*. Examination of the *urine* after the paroxysm reveals bile and an abundance of uric acid and urates. The *pulse* often becomes slowed. Exner has demonstrated the presence of about 0.4 per cent. of sugar in the urine in 39 out of 40 cases of gall-stones. On the other hand, Kausch has found glycosuria in only one of 85 cases of cholelithiasis.

The **prognosis** of biliary calculi as regards life is good, but as regards recovery only guardedly favorable. Cardiac distress with palpitation may occur during hepatic colic and form a serious complication. Fatal syncope has also been known to occur, and fatal intussusception has followed the impaction of gall-stones in the region of the ileo-cecal valve. If evidences of an infectious inflammation arise, the outlook is then more serious.

**Diagnosis.**—The diagnosis of gall-stones is sometimes extremely difficult on account of the obscure clinical symptoms and the entire absence of physical signs. When, however, the calculus becomes impacted in the duct, symptoms of biliary colic—intense pain in the epigastrium and right hypochondriac region, radiating to the back and right shoulder—usually appear. There are also fever, vomiting, and in one-half the instances jaundice and the finding of the stone in the dejecta.

**Differential Diagnosis.**—*Gastralgia* usually occurs in individuals with neurotic tendencies, and is characterized by severe paroxysmal pains in the epigastrium, extending to the back and base of the chest. It occurs often when the stomach is empty and is relieved by eating. Firm pressure over the epigastrium often alleviates the pain temporarily, and the absence of fever, jaundice, stones in the dejecta, and the negative urinalysis, together with the history of former attacks, would tend to differentiate it from hepatic colic.

**Renal Colic.**—The pain in this condition, which is often as acute as that of biliary colic, starts in the flank of the affected side and is transmitted down the ureter. The testicle and inner side of the thigh are very painful, the former being often retracted. Micturition is frequent and sometimes painful, and the urine is scanty in amount and often mixed with blood. Intense pain may also be felt in the back and abdomen, although it is usually localized in the affected side. This grouping of symptoms is wholly unlike that characterizing biliary colic.

**Intestinal Colic.**—In this variety the pain is of a boring or twisting character, usually centering about the umbilicus. It is relieved by firm pressure. Abdominal distention is often present, and relief comes with the passing of flatus. Usually there is a history of an indiscretion in



diet. When due to *lead-poisoning*, the history, the blue line on the gums, and the presence of wrist-drop would tend to confirm the diagnosis.

*Reflex colic*, due to uterine or ovarian disease, may also occur. The recurrence of the attacks, together with other symptoms pointing to disease of these organs and the exclusion of all other causes, would tend to establish the identity of the condition.

In all forms of colic, if the pain be very severe symptoms of shock may develop, indicated by vomiting, cold, clammy skin, pale and pinched features, and a rapid running pulse.

#### CHRONIC OBSTRUCTION OF THE DUCTS BY GALL-STONES.

The obstruction may exist in the *ductus choledochus*, in the cystic duct, or in both.

**1. Obstruction of the Common Duct.**—**Pathology.**—The result of the irritation produced by the presence of the stone is a catarrhal process (*cholangitis*) that may either remain chronic or terminate in suppuration (*suppurative cholangitis*). In a case of simple obstruction the gall-bladder is often moderately enlarged, though rarely extending below the lower border of the liver. The common duct is greatly distended, the stone being usually located near its termination; it is distinctly felt just beneath the mucous membrane of the descending duodenum. Occasionally two or more calculi are present, completely obliterating the canal. The hepatic duct and its branches are greatly dilated, and often contain thin, colorless mucus, the membrane lining the ducts being smooth and clear. The liver in these cases is firmer in consistency than normal, showing some increase in the connective-tissue elements (biliary cirrhosis). Following moderate enlargement of the organ progressive atrophy may rarely occur. When *suppuration* has occurred the mucous membrane is greatly swollen and reddened, and in some instances shows erosions or ulceration. The process often extends through the hepatic and cystic ducts into the liver and gall-bladder, giving rise to localized abscesses in the former and to empyema of the latter. In some instances the gall-bladder has been perforated and abscesses have formed between the liver and stomach. *Diverticula* are sometimes found post-mortem, containing biliary calculi.

**Symptoms.**—Chronic obstruction by gall-stones, with coexisting *catarrhal inflammation*, is characterized by a distinctive group of symptoms, among the most prominent of which are—

*Jaundice.*—This may be constant and very intense, or intermittent and slight, depending upon the amount of obstruction present. In some cases it disappears entirely for several months, and then recurs with varying intensity (ball-valve action of the stone). *Itching* is, as a rule, a most distressing feature.

*Pain*, occurring in paroxysmal attacks and referred to the region of the liver. This is accompanied by fever that may reach a high degree (102°–103° F.—38.8°–39.4° C.), also by chills and sweating, resembling somewhat the paroxysms of malaria. Painful points in the right side posteriorly may be annoying; these are either constant or paroxysmal.

The *chills* are often intense, and may present a quotidian, tertian, or

quartan form. The temperature of the intervals is normal. The peculiar exacerbations of temperature were first described by Charcot, and to them has been given the name of *Charcot's intermittent fever*. Many theories have been advanced as to its cause, and Murchison writes: "These paroxysms may be more or less periodic, and may extend over several months, without necessarily indicating pyemic hepatitis, the patient ultimately recovering." He further states that they are probably due to simple irritation by a stone, and are analogous to febrile paroxysms produced in passing a catheter along the urethra. Charcot believes the etiologic factor to be a septic poison, bacterial in origin and the result of chemical changes in the bile. Various micro-organisms have been detected in the bile in such cases (*bacterium coli commune*, *streptococcus pyogenes*, *et al*).

*Gastric Disturbances.*—These may be so severe during the paroxysm as to excite alarm. Intense pain is complained of in the epigastrium, accompanied often by persistent nausea and vomiting, which, however, usually subsides at the close of the paroxysm, while the jaundice at this time deepens. The attack may persist for years without progressive failure of health.

When, however, *suppurative cholangitis* occurs the prognosis becomes grave and recovery is unknown. The paroxysms occur more frequently, the fever merging into a remittent rather than an intermittent type. Grave constitutional symptoms, indicating septico-pyemia, are present, the duration is shorter, and the case rapidly tends to a fatal issue. The attacks of colicky pain occur and the jaundice, but the latter symptom is less intense than in the catarrhal form. Hepatic enlargement, on the other hand, is more marked than in the latter variety.

**2. Obstruction of the Cystic Duct.**—This almost invariably causes distention of the gall-bladder (dropsy of the gall-bladder), which may be felt distinctly below the lower edge of the liver as a pyriform, fluctuating tumor. If obstruction of the cystic duct alone occurs, *jaundice* may be entirely absent, the bile in the distended tissues being replaced by a thin, mucoid fluid. This is more apt to exist as the obstruction becomes more chronic. In some instances the distention is so great as to reach below the umbilicus, and the dilated viscus has even been mistaken for an *ovarian tumor*. Osler records a case in which 18 oz. (556.0) of fluid were removed from the gall-bladder. The contents are neutral or alkaline in reaction, albumin being often present in abundance. Catarrhal inflammation of the gall-bladder is often associated, causing *pain* and *sensitiveness* in the region of the organ. The pain may be severe and simulate biliary colic or appendicitis. The examiner can *feel* an elastic, gourd-shaped tumor closely connected with the liver, movable in respiration in the vertical, and also, under the influence of the palpating fingers, in the lateral, direction. I have observed a tongue-like projection of the anterior margin of the right lobe, to which Riedel first called attention. Given a gall-bladder well filled with stones and a relaxed abdominal wall, gall-stone crepitus may be detectable; it was felt in two cases under my immediate observation.

If the obstruction persist for a length of time, calcification or atrophy of the bladder are common sequelæ. Complete obliteration of the cavity of the gall-bladder may ensue.

Among rarer sequelæ of chronic obstruction may be mentioned—(a) *Empyema of the Gall-bladder*.—When this takes place the organ becomes greatly distended, and has been known to contain as much as a pint of purulent material. The *symptoms* of suppurative cholecystitis simulate those of purulent cholangitis, and are often preceded by those of catarrh of the gall-bladder and ducts. Perforation may occur, giving rise to circumscribed periportal abscesses or to generalized peritonitis. (b) *Phlegmonous Cholecystitis*.—This is of very rare occurrence, characterized clinically by pain and tenderness in the hepatic region, rigors and high fever, and intense prostration. It often proves fatal as the result of peritonitis from perforation.

**More Remote Effects of Gall-stones.**—These will be spoken of under three headings:

1. Stricture of the duct, resulting from ulceration and cicatrization produced by the passage of a stone.

2. Intestinal obstruction, due to impaction of gall-stones.

3. Biliary fistulæ resulting from perforations.

1. **Stricture of the Duct.**—Obliteration of the common duct may result from the passage of a gall-stone, giving rise to ulceration and cicatrization, or the stone may become impacted and lead to adhesions and permanent closure of the duct below it (Murchison). When due to ulceration the seat of the stricture is usually low down in the common duct.<sup>1</sup>

*Symptoms.*—The symptoms are those of chronic obstructive jaundice (Osler). In many cases there will be an antecedent history of the passage of gall-stones. In all cases in which the symptoms of gall-stones are followed by permanent jaundice without pain it may be suspected either that the calculus has become firmly impacted or that it has produced organic stricture or closure of the duct.

2. **Intestinal Obstruction from Impaction of Gall-stones.**—The ileum is commonly the seat of obstruction by gall-stones, that may give rise to intussusception or cause ulceration and gangrene of the bowel with perforation and fatal peritonitis. The latter event, however, occurs more frequently when the biliary concretions are situated in the cecum. Rarely they are found in the appendix, causing, as other foreign bodies, inflammatory changes, followed by ulceration and in many cases by perforation and death. Cases of impaction in the rectum of several biliary calculi have been recorded. I have recently seen a case with Dr. R. Bruce Burns.

*Symptoms.*—If the impaction occurs in the small intestine, the abdomen becomes tympanitic and tender on pressure. The contents of the stomach are first vomited, followed by bile and stercoraceous matter. Obstinate constipation persists, and symptoms of peritonitis develop and continue until either the impaction disappears or death ensues. Ileus, the result of biliary concretions, is common in females of advanced age. The history of previous acute attacks would tend to confirm the diagnosis. The pain is intense and vomiting severe and persistent. The duration of the last attack is often short, terminating fatally in a few hours.

<sup>1</sup> In vol. ix. pp. 22 and 130, *Pathologic Transactions*, two cases are recorded in which the strictures were exactly similar to those of the urethra, one being situated in the hepatic duct of the left lobe and the other in the common duct.



3. *Perforation* may occur with the establishment of *fistulous communications* between the gall-bladder and stomach, intestinal canal, bladder, vagina, lungs, abdominal parietes, or portal vein. Fistulæ between the gall-bladder and stomach are rare, though cases are recorded by Oppolzer, Frerichs, Cruveilhier, Murchison, and others. Cruveilhier states that vomited gall-stones necessarily reach the stomach through fistulous tracts, as the passage from the duodenum through the pyloric orifice would be impossible.

Fistulæ into the duodenum are of much more common occurrence, ulceration taking place usually in the fundus of the gall-bladder and in the descending or third portion of the duodenum: 39 cases are recorded of fistulous communication with the colon (Osler). I have reported a fortieth case,<sup>1</sup> which prior to reaching a fatal issue had developed widespread septico-pyemic lesions. In 6 of 9 cases reported by Murchison carcinoma of the gall-bladder was present. Fistulæ into the urinary passages may occur, 2 authenticated cases being reported. The distended gall-bladder may come in contact with the urinary viscus, or the stone may perforate into the pelvis of the kidney and pass through the ureter into the bladder.

Fistulous openings through the abdominal parietes are the most common of all fistulæ, the place of exit of the biliary concretions being usually in the region of the gall-bladder or at the umbilicus, to which (according to Murchison) it may be directed by the suspensory ligament of the liver. As many as 600 stones have been removed from the gall-bladder in this manner. They vary greatly in size, being often as large as a goose-egg. Advanced life and female sex are said to be predisposing causes. Murchison records 5, and Courvoisier's statistics show 184 cases, in 78 of which recovery took place.

Fistulæ into the pleura, bronchi, and vagina have been recorded, but are extremely rare. Courvoisier records 24 cases of fistulæ into the lungs, only 7 of which terminated in recovery. Fauconneau, Dufoesne, Frerichs, Bristowe, and Murchison mention cases of fistulæ into the portal vein, with the presence of biliary concretions in the latter.

**Diagnosis.**—I would strongly urge an exploratory celiotomy as an accurate means of diagnosis in obscure cases.

**Treatment of Foregoing Conditions.**—The indications for treatment in *cholelithiasis* are (*a*) to remove the cause; (*b*) to relieve the paroxysms of hepatic colic; and (*c*) to adopt palliative or radical measures for the removal of the stones.

**Preventive Treatment.**—In this, as in the treatment of jaundice, diet and hygiene play an important part. The former should be as simple as possible, consisting largely of skimmed-milk, lean meat, eggs, fruit, and green vegetables. Fatty foods, sugars, starches, and pastries are to be strongly interdicted. All foods should be thoroughly masticated, so as to digest easily, and meals should be taken at regular intervals. Systematic exercise in the open air is of signal value, as it stimulates the flow of bile. Punkhauer strongly recommends horseback-riding, believing this to be efficient in removing obstructions in the common duct.

Among the drugs mostly used in the treatment of this condition I would advise the following: Sodium sulphate, combined with the extract of taraxacum (Harley); ox-gall (Dubney), in 5- to 10-gr. (0.324–0.648)

<sup>1</sup> *Clinical Lecture, International Clinics*, vol. ii. third series, p. 27.

doses, three times daily (to relieve flatulency and stimulate the biliary secretion); sodium salicylate (gr. x to xv—0.648 to 0.972, three times daily); and sodium chlorate (gr. iv to vj—0.259 to 0.388) three times a day (Schiff).

The bowels should be kept freely soluble, constipation being carefully avoided. In my own experience a dram (4.0) of sodium phosphate or of Rochelle salts in concentrated solution in the morning on rising has yielded excellent results. Other laxatives whose use is to be advised and encouraged are cascara sagrada, podophyllin, and rhubarb.

**Treatment of the Paroxysm of Biliary Colic.**—At the very onset of an attack of hepatic colic the prompt exhibition of morphin or of codein may greatly mitigate an attack. The former may be given hypodermically in  $\frac{1}{8}$  to  $\frac{1}{4}$ -gr. (0.008–0.016) doses every hour until relief follows; the latter is exhibited by the mouth in doses of 1 gr. (0.0648) every hour. Inhalations of chloroform, with morphin hypodermically, the former being continued until the latter has taken effect, may be regarded as the typical treatment during an attack.

Hot baths and hot applications (with counter-irritation) over the liver are valuable aids in the treatment of hepatic colic, being given at a temperature of 98° to 100° F. (36.6° to 37.7° C.), and continued for twenty minutes if endurable, so as to effect relaxation. If cardiac depression results and the pulse becomes weak, the baths should be discontinued. Hot flaxseed-poultices, cloths wrung out of hot water, hot hop-bags, or turpentine stupes may be applied over the hepatic region until the attack subsides. Ice-poultices have been advised by Buchetan.

If shock or syncope should develop, the body-temperature must be maintained by hot bottles or bricks placed in contact with the surface of the body, together with strychnin (gr.  $\frac{1}{30}$ —0.0021), atropin (gr.  $\frac{1}{150}$ —0.00042), and brandy (1 dram—4.0) hypodermically.

*Nausea and vomiting* may be reduced by 15-drop doses of spirits of chloroform every half hour; also by brandy and soda-water or champagne.

In mild cases sodium salicylate (gr. viij–xv—0.518–0.972 in twenty-four hours), recommended by Prevost and Binet, or codein (gr. j), with phenacetin (gr. x), every few hours gives relief. The free use of olive oil or glycerin in hepatic colic has been followed by a beneficial effect (Rosenberg, Goodhart). The former is given in quantities of 4 to 6 oz. (128.0–192.0) by the mouth every three or four hours, nausea being prevented by concealing the taste with lemon-juice; the latter, recommended by Ferrand, is given in doses ranging from 1 to 2 tablespoonfuls, repeated in the same length of time. Both remedies are supposed to do good by increasing the flow of bile, thus forcing the stone outward toward the bowel.

**Treatment for Removal of Gall-stones.**—The palliative treatment consists in the administration of agents that tend to increase the flow of bile. The free use of pure water by the mouth, together with copious rectal injections daily of cold water, has been found effective. It may be rendered alkaline by sodium bicarbonate or borate in a 3 per cent. solution.

A course of alkaline treatment at some of the more noted mineral springs (Bedford, Vichy, Carlsbad) is often attended with good re-

sults. Perhaps the three best cholagogues that may be mentioned are sodium phosphate, sodium cholate, and ox-gall. Olive oil and glycerin also increase the secretion of bile.

Willoughby reports a case in which prompt recovery ensued from the use of toluylenediamine after three years of unsuccessful treatment; he began with 1 grain daily, and increased to 2 grains.

Agents to dissolve the stone have been tried at various times, among them being *Durande's method* (turpentine and ether), but, so far, all such methods of treatment have been unsuccessful.

Of the various surgical measures for the removal of gall-stones the following are the chief: (a) Removal of the stone from the common duct (choledochotomy); (b) Removal of the stone from the cystic duct (cholecystotomy); (c) Establishing a fistulous opening between the gall-bladder and the bowel (cholecystenterostomy); (d) Extirpation of the gall-bladder (cholecystectomy), the latter operation giving a mortality of 17 per cent., according to Murphy's statistics. And operative procedure is indicated in infectious (suppurative) cholecystitis as well as in infectious (suppurative) cholangitis.

## CARCINOMA OF THE BILE-DUCTS.

THE biliary passages may be the seat of carcinoma, which may occur primarily and exist over a long period of time without being recognized.

**Pathology.**—The gall-bladder, as the result of obstruction of the duct, is often greatly distended, measuring as much as 7 inches (17.7 cm.) in length (in a case reported by Harley) from the entrance of the duct to the fundus, and being filled with a cloudy liquid, somewhat resembling barley-water, that contains flakes of epithelium, granular matter, and particles of inspissated bile. If the growth be near the duodenal orifice, the common and cystic ducts are often greatly distended, and the dilatation may extend into the hepatic ducts and their branches. The liver may be enlarged, and in some instances presents the secondary nodules that are characteristic of the disease. Microscopically, carcinoma of the gall-bladder exhibits marked variations in different cases; "it may be either columnar or spheroidal-celled" (Rolleston).

**Etiology.**—The causes of carcinoma of the bile-ducts are the same here as elsewhere, and among these the mechanical or inflammatory theory of Virchow must be accepted. Tight-lacing and mechanical irritation by gall-stones are followed in many instances by cancerous degeneration; Osler states that "biliary calculi are present in at least seven-eighths of all cases." Among other factors, heredity and age (after forty) play an important part. Although carcinoma of the *liver* undoubtedly occurs more frequently in males, Musser found that out of 100 cases of carcinoma of the *ducts*, 75 were female; and Ames found the ratio to be 4 to 1 in favor of females.



**Symptoms.**—The signs and symptoms, according to Harley, present nothing characteristic to distinguish them from other causes of obstruction in the ducts. On *palpation* in the early stages the gall-bladder is found moderately enlarged, but later it rapidly undergoes diminution in size. *Jaundice* becomes very intense, and remains permanent. Throughout the course of the disease all the symptoms referable to chronic obstruction of the duct by gall-stones (paroxysmal pain, gastric disturbance, rise of temperature, Charcot's fever) may develop.

Examination of the urine and feces reveals the presence of *bile-pigment* in the former and its absence in the latter. The urine often shows the presence of bile-stained casts (*vide* Fig. 63).

*Ascites* not rarely occurs during the later stages, with the involvement of surrounding organs by contiguity, as well as with the appearance of secondary nodules in the liver and the development of cachexia.

**Diagnosis.**—Carcinoma of the biliary ducts cannot always be detected by physical examination. Distinct evidence of chronic obstruction of the duct, as persistent and intense jaundice (which occurs in three-fourths of the cases), the development of cachexia and the absence of cancerous involvement of other organs, however, will tend to characterize it. Often a hard tumor-mass is present in the region of the gall-bladder, projecting in the direction of the umbilicus. It should be recollected that the bile-ducts are oftener the seat of the primary affection than the liver. An assured diagnosis, however, is often impossible.

**Prognosis.**—The prognosis of carcinoma of the bile-ducts is, like that of other organs, absolutely fatal, though the course of the disease is not so rapid as that of carcinoma elsewhere until secondary involvement of the liver occurs.

**Treatment.**—The treatment is merely palliative. Operative measures are rarely justifiable, since the disease is rarely recognized before the liver becomes involved. As seven-eighths of the cases follow obstruction of the duct by gall-stones, the preventive treatment of the latter should be carefully observed whenever symptoms of disordered liver-function manifest themselves.

The treatment of the pain, anemia, and emaciation will be described in the discussion of Carcinoma of the Liver (*vide* p. 899).

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## STENOSIS OF THE BILE-DUCTS.

STENOSIS may result from any of the following causes: (*a*) *Round-worms* in the duct (rarely); (*b*) Foreign bodies, as seeds; (*c*) Ulceration and cicatrization following the passage of gall-stones (most commonly); (*d*) Pressure from without, as from tumors (carcinoma chiefly) of the head of the pancreas and pylorus (rare); (*e*) Abdominal tumors; (*f*) Aneurysm of the abdominal aorta or of the celiac axis (rare); (*g*) Secondary enlargement of the lymphatics of the liver (common); (*h*) More rarely in man than in the lower animals distoma hepaticum of liver-flukes and echinococci; (*i*) Adhesions due to chronic peritonitis.

**Pathology.**—If the stenosis is of recent origin, the liver is enlarged

and shows more or less congestion, with some increase of the connective-tissue elements. The substance is firmer than normal, the color varying from an olive-green to a deep bronze. If, however, the obstruction be of long standing, the presence of the dilated intra-hepatic ducts and the increase of connective tissue cause secondary atrophy of the hepatic cells, with a diminution in the size of the organ.

**Symptoms.**—The symptoms vary greatly according to the cause of the stenosis, but in the main they are those of chronic obstruction of the duct—viz. paroxysmal pain in the region of the liver, referred to the right shoulder; jaundice of varying intensity, but gradually deepening after each attack; and gastric disturbance, with ague-like paroxysms (fever and sweating), the latter being most frequently met with in occlusion from gall-stones.

**Diagnosis.**—The pathognomonic symptoms determining the nature of the stenosis are very often wanting, and the diagnosis is rendered correspondingly difficult. On the other hand, stenosis or complete occlusion of the bile-passages calls for diagnosis principally on account of the special cause or causes of the given case.

When the condition is due to *lumbricoid* worms reflex symptoms usually appear, as pruritus of the nose and anus, grinding of the teeth during sleep, and convulsions.

In *carcinoma of the head of the pancreas or the pylorus* pressing on the ducts the growth may be detected by palpation, together with a recognition of other more or less characteristic features (*vide infra*, p. 911), and the rapid course of the disease.

*Abdominal aneurysm* may give rise to obstruction of the duct without being evidenced by physical signs. Usually, however, when the sacculatation presses against the bile-duct, the throbbing in the epigastrium, the tumor (which can often be grasped), and the expansile pulsation on palpation will tend to establish the cause of the obstruction.

When due to *cancerous nodules in the liver* there is usually a history of primary carcinoma of the stomach, mammary gland, rectum, or of one of the pelvic viscera. Osler records a case in which jaundice (thought to have been catarrhal in origin) developed seven weeks previously. On careful examination "a small nodule was detected at the umbilicus, which on removal proved to be scirrhus."

When the stenosis is due to *ulceration* following the passage of gall-stones, the history of biliary colic and of the presence of calculi in the dejecta, and the paroxysmal pain with jaundice and intermittent fever, will serve to establish the cause.

If the fever be of the continued type and the liver uniformly enlarged, with the development of jaundice, the case is probably one of *hypertrophic cirrhosis*; whereas if the enlargement be progressive and nodules can be detected on palpation in addition to the appearance of cachexia and jaundice, *carcinoma* is undoubtedly present.

**Physical signs** aid but little in the diagnosis, as obstruction of the common duct is usually unattended by any great enlargement of the gall-bladder.

In many cases only by remembering the various causes and eliminating them carefully, one by one, can the diagnosis be positively made.

**Prognosis.**—It may be said of the prognosis, as of the symptoms, that both vary according to the cause of the stenosis. Generally speaking, the outlook is rather grave, since many of the causative conditions are fatal. If the obstruction is due to cicatricial contraction, the prognosis is guardedly favorable as to life, but hopeless as to recovery. If the obstruction is permanent, the case ends fatally.

**Treatment.**—The treatment of occlusion of the bile-ducts varies according as it is due to cicatricial contraction following ulceration or to foreign bodies (seeds or lumbricoid worms), or to gall-stones or tumors pressing upon or involving the ducts or adjacent organs (pancreas, pylorus). If the stenosis follows ulceration in the duct, and is sufficient to cause almost complete occlusion with biliary retention, the operation of cholecystenterostomy may become necessary in order to prevent dilatation of the gall-bladder with resorption of bile.

Foreign bodies in the duct may be removed by free purging, aided by the liberal use of alkaline mineral waters. In critical cases the operation of cholecystotomy has been practised.

Gall-stones form the most frequent cause of stenosis, and the treatment, both for the prevention and removal of calculi, has already been described in the discussion of Biliary Calculi (*vide* p. 859).

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### ICTERUS NEONATORUM.

**Definition.**—Jaundice occurring in the new-born. This may be either *pathologic* or *physiologic*—a slight tinting of the skin occurring quite commonly in the new-born.

**Pathology.**—The morbid anatomy of the *pathologic* form varies with the cause of the jaundice. The secretion of bile, like the secretion of urine, begins long before birth, and Zweifel has found bile-pigment and bile-acids in the contents of the intestines of a three-months' fetus. Hence children may be born laboring under an attack of well-marked jaundice.

In well-marked cases of pathologic jaundice the skin presents a deep greenish-yellow hue. The internal tissues are also stained. Knopfmacher has studied the condition of the blood, and found that the red cells presented no signs of destruction, but rather of active new-formation.

**Etiology.**—Of the *physiologic* forms, the following are the main causes: 1. The *ductus venosus* may remain patulous, allowing some of the portal blood, containing bile, to flow into the systemic circulation (Quinke). 2. Diminished pressure in the portal vessels from ligation of the umbilical vein causes increased tension in the hepatic capillaries and absorption of bile.

**Pathologic Icterus.**—The causes are the following: (*a*) Congenital stricture or absence of the duct; (*b*) Syphilitic disease of the liver; (*c*) Septic processes set up by infection through the umbilical vein.

**Symptoms.**—In *physiologic* jaundice the skin is tinted greenish-



yellow, resembling somewhat that of chlorosis. The mucous membranes are pale and the conjunctivæ pearly-white. The pulse is feeble and sometimes rapid. Auscultation over the base of the heart often reveals a soft systolic murmur transmitted to the vessels of the neck and associated with a venous hum. According to Murchison, false or physiologic jaundice differs from the true or pathologic form in that—1. The conjunctivæ are of a natural color; 2. The urine is free from bile-pigment; 3. The yellow color gradually fades from the skin after a few days; 4. The child is quite well and the bowels are acting properly.

In *pathologic* jaundice the skin and conjunctivæ are more or less intensely icteroid, the urine is loaded with bile-pigment, while the feces are of the pipe-clay variety. Hemorrhage from the cord may occur and destruction of life may be rapidly accomplished, or the condition may last for some weeks without serious impairment of the general health, with final recovery.

**Treatment.**—In the milder cases calomel in minute doses, combined with lactopeptin and sodium bicarbonate, can be recommended. In malignant cases treatment is of no avail.

## VASCULAR (CIRCULATORY) AFFECTIONS OF THE LIVER.

### ANEMIA.

THE physical symptoms of this condition are absolutely *nil*, and its existence only discoverable *postmortem*. Its most common causes are those of general anemia, fatty and amyloid degeneration.

### HYPEREMIA.

**Definition.**—An excess of blood in the liver. This may be of two varieties: (*a*) *active* and (*b*) *passive*, the latter being the more common.

#### ACUTE HYPEREMIA.

(*Active Congestion.*)

**Definition.**—An excess of arterial blood in the liver.

**Etiology.**—Among the common causes are rich living, sedentary habits, alcoholism, traumatism, acute infectious diseases (typhus, typhoid), and pernicious malaria. The condition may also be vicarious, due to a sudden cessation of menstruation or of hemorrhage in other parts of the body. A *physiologic* condition is the temporary hyperemia that occurs during the ingestion of a full meal.

**Symptoms.**—There are no symptoms characteristic of this condition; those present in the different cases are varied and referable to disturbances of other viscera, as in coexisting cardiac hypertrophy or gastrointestinal catarrh. Often, however, there is a sense of *fullness* and *distress* in the right hypochondrium after eating, with *tenderness* on palpation over the lower margin of the organ.

**Prognosis and Course.**—It is impossible to make any definite statement as to the course and prognosis of active hyperemia, these depending

wholly upon the cause of the affection. When due to errors of diet and hygiene the condition is easily remedied; the prognosis of hyperemia accompanying hepatic cirrhosis, however, is decidedly grave.

#### PASSIVE HYPEREMIA.

(*Passive Congestion.*)

**Definition.**—An increase of venous blood in the liver.

**Pathology.**—The organ is enlarged in size and changed to a deep-red color, its substance being firmer than normal. The center of the lobule (the area of the hepatic vein) becomes deeply pigmented, the periphery (occupied by the portal vein) being lighter in color, sometimes owing to fatty infiltration. Because of its mottled appearance this has received the name of the “nutmeg liver.”

In long-standing passive congestion there is an increase of connective tissue, due to a proliferation of round-cells, causing atrophy of the parenchyma. The blood in the central capillaries becomes altered, the capillaries themselves are distended, and brown pigment is deposited about the center of the lobules. The organ becomes very much darker in color, and to this condition the name “cyanotic induration” or “cardiac liver” has been given. Later, contraction of the connective tissue occurs, causing a diminution in the size of the organ, and forming the so-called “atrophic nutmeg liver.”

**Etiology.**—The causes that lead to passive hyperemia are both *local* and *general*. Among *local* causes may be mentioned the following:

1. Pressure over the portal area from without, as from a tumor or cyst.
2. Disease of the walls of the veins, as in syphilitic phlebitis.
3. Coagulation of the blood in the veins (thrombosis).

Among the *general* causes are—

1. Chronic valvular disease affecting the right side. Passive hyperemia is also common in mitral disease.
2. Pulmonary emphysema and cirrhosis of the lung.
3. Intrathoracic tumors, which by their mechanical action cause an increased pressure in the efferent branches of the hepatic veins.

**Symptoms.**—Often the patient experiences a sensation of *fullness* and *weight* in the region of the liver that amounts in some instances to actual *pain*. *Jaundice* is usually present, but varies in intensity, and is due to obstruction of the smaller ducts by the distention of the hepatic venules. *Hematemesis* is not rare, and symptoms of gastro-intestinal disturbance are usually present. In marked cases the *stools* are *clay-colored*, showing the absence of bile; the *urine* is loaded with bile-pigment; and jaundice deepens with the development of *ascites* or *anasarca* from portal obstruction. On *palpation* the organ is tender and increased in size, extending in some instances fully a hand's breadth below the costal margin. In marked cases the whole organ pulsates, owing to the regurgitation of blood into the hepatic veins. This symptom is best elicited by placing one hand on the ensiform cartilage, while the other presses against the liver below the right lower border of the ribs.

**Diagnosis.**—The diagnosis of passive congestion, *per se*, is often very difficult, but when secondary to heart- and lung-diseases it is rendered more plain.

The **prognosis** and **treatment** depend wholly upon the causal factors.

## DISEASES OF THE PORTAL VEIN.

### THROMBOSIS AND EMBOLISM.

**Pathology.**—In the early stages the clot presents a grayish-red or yellowish appearance, and on loosening it is found to adhere more or less closely to the inner coat of the vein. Later it becomes a mass of small white fibrin tightly adherent to the sides of the blood-vessel, which itself undergoes fibroid change, giving rise to the so-called adhesive pyelephlebitis. Organized thrombi are rarely found, except in the smaller branches of the portal area. If the thrombus obstruct the vessel, collateral circulation may be established for years, as in a case recorded by Osler. Septic softening, however, is a very common result, and most frequent of all is pyelephlebitis. If a parietal or channelled thrombus be formed, partial or complete circulation may be re-established and recovery take place. Hemorrhagic infarction may take place, but is very rare.

**Etiology.**—*Thrombi* are rare occurrences in the portal vein. Among the *causes* that lead to their occurrence, however, may be mentioned—(a) Traumatism; (b) cirrhosis; (c) carcinoma of the liver, involving the portal area; (d) pressure from without, as in proliferative peritonitis involving the gastro-hepatic omentum, abscesses, enlarged glands, or impacted calculi pressing on the veins; (e) it may be occasioned by ulcerative affections of the bowels and appendicitis, and pyelephlebitis may precede its occurrence; (f) slowing of the circulation due to splenic diseases, such as marasmus.

**Symptoms.**—Symptoms may be almost lacking in portal obstruction, or the condition may simulate cirrhosis of the liver. In ordinary cases the symptoms are very slight, the hepatic circulation, as shown by Cohnheim and Litton, being “sufficient for the nourishment of the liver and secretion of the bile” (Henry).

If the occlusion be *complete*, *edema* followed by the rapid development of *ascites* may occur. In such cases loss of strength is persistent and progressive, and death may result from exhaustion. *Hemorrhages* due to venous stasis may occur from the nose, stomach, and intestines. *Jaundice* and *diarrhea* occur frequently, the former being the result of obstruction to the biliary passages from the same causes that produce the thrombosis or the diminished pressure in the portal area. On *palpation* the liver is found slightly enlarged and tender on pressure, and projecting below the lower margin of the ribs; the *spleen* is also enlarged. *Percussion* also reveals enlargement over the splenic area. If ascites is present, percussion will reveal dulness in the flanks, changing with the position of the patient; and on gently tapping one side of the belly-wall, with the hand on the opposite side, a wave of fluctuation will be felt.

**Diagnosis.**—The diagnosis of portal thrombosis is often extremely difficult. “A suggestive symptom, however, is sudden onset of the most intense engorgement of the branches of the portal system” (Osler).

**Sequelæ.**—If the emboli are septic in origin, an abscess, with all its accompanying symptoms, will be the result. Hemorrhagic infarction



may occur, but is very rare, since a free anastomosis exists between the lobular plexuses and the hepatic artery.

“Pylethrombosis may be regarded as probable if no other possible cause of the portal obstruction seems likely, and if we are able to discover a cause for thrombosis, like a former attack of circumscribed peritonitis” (Strümpell).

The **prognosis** is always unfavorable, although certain cases have been demonstrated by autopsy to have improved temporarily.

**Course and Duration.**—Nothing definite can be stated in regard to the course and duration of this affection, since these depend entirely upon the cause.

**Treatment.**—The symptoms resulting from portal congestion, due to thrombi in the portal vein, are those described under Cirrhosis of the Liver, and the treatment is identical with that of interstitial hepatitis. In rare instances septic emboli give rise to abscesses that are usually multiple; when these occur the treatment is purely symptomatic.

#### SUPPURATIVE PYLEPHLEBITIS.

**Definition.**—A purulent inflammation of the portal vein or its branches.

**Pathology.**—If noted in the early stages, the coats of the portal vein are distended and thickened, and the connective tissue surrounding the portal area is infiltrated and the seat of minute ecchymoses. The inflammation usually originates in the smaller veins of the portal system or in the hepatic branches of the vein itself; the main trunk is attacked least often. Numerous thrombi are found obstructing the vein and its branches, which finally undergo suppuration. From these, emboli enter the circulation and are carried to all parts of the liver, forming metastatic abscesses. In advanced cases the whole organ (especially the peripheral parts) becomes infiltrated with pockets of pus, that communicate with the portal vein or its branches, and extend in some instances into the mesenteric or gastric veins. A single large abscess may be present, but multiple abscesses are the rule. The contents may be very fetid and bile-stained, or, as in many instances, they may be composed of thick, creamy laudable pus. From this focus of suppuration embolic abscesses may extend to the lungs, brain, kidneys, and joints.

The macroscopic appearance, with the organ *in situ*, is sometimes practically normal. The liver may present a uniform enlargement, the surface being of normal color and the capsule non-adherent. More commonly, however, the cortex presents a mottled appearance, and numerous yellowish-white spots are seen beneath the capsule.

**Etiology.**—The most frequent source of purulent pylephlebitis is appendicitis with abscess. Rarely the disease arises idiopathically.

Among other causes are the following: (*a*) A secondary (becoming a general) pyemia. (*b*) Ulceration of the intestines, occurring in dysentery and, more rarely, in typhoid fever. (*c*) Gastric ulcer. (*d*) Pelvic abscess; abscess of the spleen. (*e*) Specific infection through the umbilicus, occurring in the new-born.

**Symptoms.**—The symptoms vary according as to whether the case remains one of suppurative pylephlebitis or terminates in hepatic ab-

scess. If the condition is part of a general *pyemia*, the symptoms referable to the liver may be almost negative. The *liver* is usually enlarged, and tender on pressure, the enlargement being most marked when an hepatic abscess exists. Though *pain* is present, in many cases it is not a marked feature; it is frequently referred to the epigastrium, and may radiate laterally or downward. *Percussion* in the left axillary line shows splenic enlargement, and the organ can in some instances be felt below the costal margin, constituting the "*acute splenic tumor*" of septico-pyemia.

The *fever* is of an irregular septic type, the elevation in temperature is accompanied by rigors or chills and followed by profuse sweating. Other febrile symptoms, as headache, anorexia, and scanty, high-colored urine, are present. *Jaundice* of varying intensity is present, although usually it is not pronounced, the complexion being merely doughy or muddy. *Diarrhea* is not an infrequent symptom of this condition, and the dejecta sometimes contain blood as a result of the venous engorgement. Nausea and vomiting are often marked. As the case advances the pulse becomes rapid and small, and a low form of delirium develops; this is followed by stupor, coma, and death.

**Duration and Prognosis.**—The duration of suppurative pylephlebitis is usually from one to three or four weeks or longer. The prognosis is absolutely fatal.

**Diagnosis.**—The diagnosis of suppurative pylephlebitis is sometimes extremely difficult, unless the case is complicated by hepatic abscess, as enlargement of the liver is not constant in the former condition. The etiology, septic temperature, enlargement of the spleen, jaundice, and pain in the region of the liver would all, however, point to this affection.

The *differential diagnosis* of hepatic abscess will be spoken of later.

**Treatment.**—Unfortunately, the treatment of suppurative pylephlebitis can only be palliative. Surgical measures are rarely curative, unless the abscess is single and localized and shows signs of pointing. Free stimulation should be begun early, and should be persisted in throughout the course of the disease.

*Nausea and vomiting* may often be relieved or controlled by pellets of cracked ice, brandy, and soda-water or champagne. One-drop doses of wine of ipecac every half hour until relieved, or the antiemetics, as creasote ( $M\frac{1}{2}$ —0.033—every half hour combined with bismuth subnitrate gr. v—0.324) or cerium oxalate (gr.  $\frac{1}{4}$ —0.016—every two hours), often check the gastric irritability.

The *pain* in suppurative pylephlebitis may be acute, and demand the free use of morphin, either hypodermically or by the mouth. If much nausea exists, suppositories containing the extract of opium may be given at intervals. As the disease is almost invariably fatal, opium or its alkaloids may be given liberally.

*Fever* may be controlled by repeated cold spongings or by the cold pack (68° F.—20° C.), aided by large doses of quinin and salol. As the pyrexia is pyemic in character, however, drugs have little or no effect in reducing the temperature.

*Delirium*, which, with the rise of temperature, usually becomes aggravated toward evening, can best be subdued by applying an ice-cap to the head; this may also be reinforced by motor and sensory depressants,

as chloral hydrate and the bromids. In well-marked cases hypodermic injections of hyoscin hydrobromate (gr.  $\frac{1}{100}$ —0.0006—every two hours until relieved) may be necessary.

#### STENOSIS.

Obstruction of the portal vein may be due, as before mentioned, to (*a*) thrombosis; (*b*) cicatricial contraction from cirrhosis or syphilis of the liver; and (*c*) tumors pressing on the portal area. The first cause is the more common, chiefly because mechanical obstruction, by causing a stasis of the blood-current, induces the formation of a thrombus.

The **symptoms** of portal stenosis may be *nil*; if the stenosis occurs slowly, the hepatic artery furnishes sufficient blood to carry on the functions of the liver, the compensatory circulation being established by means of the systemic vessels. If due to thrombosis, the symptoms of portal engorgement appear suddenly with the development of edema and ascites. The liver is rarely enlarged in this condition.

**Prognosis.**—This depends wholly upon the cause of the affection. Thrombi in the portal vein often give rise to a suppurative pyelephlebitis, terminating in hepatic abscess; tumors are rarely accessible; whereas fibroid conditions of the liver causing cicatricial contraction are incurable. As a rule, the prognosis may be said to be guardedly unfavorable.

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### AFFECTIONS OF THE HEPATIC BLOOD-VESSELS.

OSLER records a case of *stenosis of the hepatic veins* that was associated with fibroid obliteration of the inferior vena cava, with a greatly enlarged and cirrhotic liver.

Among other affections of the hepatic veins are (*a*) Emboli, originating from a thrombus in the right auricle, and (*b*) Dilatation, from stasis of the blood-current flowing to the right heart, due to enlargement of the latter.

Affections of the *hepatic arteries* are exceedingly rare, but may occur in one of the following forms: (*a*) *Aneurysm*.—Only 10 or 12 cases of aneurysm have been reported. (*b*) *Hypertrophy and Dilatation*.—These may occur in connection with general hepatic cirrhosis, the cicatricial bands obstructing the lumen of the artery, and causing thickening in some places, and ampullæ, or sac-like dilatations, in others. (*c*) *Sclerosis*.—This may form a part of a general arterio-sclerosis, though it occurs oftener in connection with cirrhosis or syphilitic hepatitis.

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### ATROPHY AND HYPERTROPHY OF THE LIVER.

(*a*) *Atrophy*.—Simple atrophy of the liver may result from pressure (corset-liver), syphilis, advanced cirrhosis, senility, and from the toxic



action of phosphorus, arsenic, or chloroform—all factors that induce rapid fatty degeneration with cell-destruction.

(b) *Hypertrophy* is of two kinds—(1) *true* and (2) *false*. (1) *True hypertrophy* may be subdivided into *simple* and *numerical* (hyperplasia), the latter referring to an increase in the number of the parenchymatous cells, and not, necessarily, implying an increase in the size of the organ.

The two causes of simple hypertrophy are active and passive congestion. Among the causes of numerical hypertrophy may be mentioned the following: Leukemia, hypertrophic cirrhosis, atrophic cirrhosis (hyperplasia), syphilis, diabetes, and malaria.

(2) *Pseudo- or false hypertrophy* occurs in amyloid and fatty infiltration, carcinoma, and abscess, and consists in an increase in the tissues least concerned in the function of the organ.

## HEPATIC INFILTRATIONS AND DEGENERATIONS.

### AMYLOID INFILTRATION.

(*Waxy, Lardaceous, Bacony, or Albuminoid Infiltration; Amyloid Disease.*)

**Definition.**—A deposit in the hepatic connective tissues of a peculiar substance having some of the reactions of, and resembling, starch. A physiologic example of amyloid infiltration may be found in the *corpora amylacea* of the prostate gland, in which there is a concentric arrangement somewhat resembling a starch-granule.

**Pathology.**—The organ is larger than normally and of firmer consistence. The edges are rounded and not well defined, and the surface is of a light color, presenting in some instances a mottled appearance. On section the surface presents a grayish-brown, glistening appearance, which when scraped fails to exude oil-droplets, as in the fatty liver.

On microscopic examination the connective-tissue trabeculae and the intima and media of the capillary walls (the starting-points) are chiefly affected, the lumen of the latter being lessened; this decreases the blood-supply to the liver, and often directly induces fatty degeneration. The hepatic cells may be atrophied and show evidences of fatty change. Amyloid material is structureless, and appears in small cloudy masses under the microscope. Chemically, it contains small amounts of potassium and phosphorus and an excess of sodium and chlorin.

**Etiology.**—Amyloid infiltration may occur primarily in the liver, but it is often a part of a general infiltration, affecting especially the spleen (*sago spleen*) and kidneys. It is also found in some syphilitic scars and in certain tumors and old thrombi.

Dickinson believes that the deposition of amyloid material is due to a decrease in the alkalinity of the fluid of the body, the pus (in cases of long suppuration) having removed a large quantity of the natural potassium salts. In malarial cachexia, however, such losses could not have occurred.

It is a frequent sequel to long-standing and exhausting suppurating and cachectic affections, as necrosis of the bones, hip-joint disease, and

pyelitis; "especially is this the case when they occur in an hereditary tuberculous or syphilitic constitution" (Harley). Amyloid disease may also complicate chronic malaria. In children tuberculosis and rachitis not uncommonly contribute to amyloid infiltration.

*Tests and Characteristics of Amyloid Material.*—Although of animal origin, amyloid matter is closely related to a vegetable albuminoid starch. It is, however, slow to decompose, and is not acted on by weak acids and alkalies, whereas strong alkalies dissolve it. Iodin gives a blue color upon the addition of sulphuric acid. Lugol's solution (the aqueous solution of iodine and potassium iodide) gives a brown tint to amyloid liver-substance and stains ordinary hepatic tissues a yellow color. Gentian-violet gives a reddish or pinkish hue to amyloid substance, while normal tissue is stained blue.

The following is taken from Harley's *Comparative Table of Amyloid Tests*:

	STARCH.	AMYLOID.	CHOLESTERIN.
Water.	Dissolves on boiling.	Dissolves on boiling.	Unchanged.
Ether.	Insoluble.	Insoluble.	Dissolves.
Heat.	Dries up.	Dries up.	Melts.
Sulphuric acid.	Chars.	Swells up, reddish-brown.	Becomes green, blue, etc.
Iodin.	Becomes blue.	Blue color with $\text{H}_2\text{SO}_4$ , which is destroyed by excess.	Remains unchanged.
Sulphate of indigo. . . . .		Amyloid tissue soaked in it becomes a brilliant blue, while with ordinary liver-tissues the blue fades to a pale green.	

**Symptoms.**—When amyloid disease occurs in *children* the subjects are poorly developed and puny, the complexion is, as a rule, muddy or sallow, and the abdomen usually prominent. Occasionally the *skin* is exceedingly transparent. At any age *gastro-intestinal symptoms* occur, prominent among which are marked constipation and a capricious appetite. *Mental phenomena*, as impairment of memory and inability to concentrate, are not unusual in this disease. *Pain* about the hepatic region is a rare symptom. The *spleen* is usually enlarged from coexistent amyloid infiltration. The *urine* often contains albumin (globulin is nearly always present) and waxy tube-casts; it is of high specific gravity, and is usually scanty and dark colored. The *physical signs* show an increase in the area of hepatic dulness; the edges of the organ extend below the costal margin and have a rounded outline. Sometimes, however, the edge, even in a very great enlargement, is sharp and large. Wilks speaks of an amyloid liver weighing 14 lbs.—6.35 kgms. (Ösler). In rare instances the liver is reduced in size.

**Diagnosis.**—The foregoing symptoms and physical signs, in conjunction with an ordinarily clear etiology, are sufficient to establish the diagnosis.

**Treatment.**—As amyloid disease is almost invariably a secondary

condition, the treatment must be directed to the removal of the primary cause, whether syphilis, tuberculosis, or rickets. The diet should consist of nitrogenous or animal substances, with a minimum amount of fatty or farinaceous foods. French rolls and bran- or gluten-bread are allowable, together with lean meat and green vegetables. Stimulants are to be strictly avoided. Moderate exercise, with the judicious use of Turkish (hot-air) and Russian (hot-vapor) baths, is also of great value.

Many drugs are mentioned in the treatment of this disease, among the more important being the ammonium salts (the chlorid, gr. v to x—0.324 to 0.648—three or four times a day), and other alkalies, together with tonics and laxatives.

When syphilis has been clearly established as an etiologic factor of the disease, the tincture of iodine in 10- to 15-minim (0.666–0.999) doses, well diluted, has been recommended to be given three or four times daily. Cod-liver oil as a nutritive has been tried with good effect. Of tonics, the dilute mineral acids, given in moderate doses over a long period of time, have probably achieved the best results.

#### FATTY INFILTRATION.

**Definition.**—A deposit of fat in the hepatic tissues due to the ingestion of fats and albuminates.

**Pathology.**—The infiltration occurs often in localized areas, and may be so intense that the organ when cut presents a shiny, oily appearance. The liver is often evenly enlarged, and may weigh twelve to fifteen pounds. The edges are rounded and the substance less firm than normally. Portions of the liver-substance float in water, being of low specific gravity. The color is light-yellow or grayish. On microscopic examination the protoplasm of the cell is seen to be pushed to one side by the fat-droplets, which tend to coalesce. When the fat is removed the cells resume their normal outline and appearance.

**Etiology.**—(a) Fatty infiltration may form part of a general obesity or it may follow excessive over-eating or sedentary habits. (b) It often occurs in wasting diseases, as carcinoma, syphilis, chronic malaria, and tuberculosis, and often accompanies fatty degeneration.

**Symptoms.**—The subjective symptoms of fatty infiltration may be entirely wanting, since the function of the liver is not impaired to any extent. When they are present progressive *anemia* and *debility* are noted, and are accompanied by *nervous irritability* and *insomnia*. In marked cases the cardiac rhythm is disturbed, causing a *feeble* and *irregular impulse*.

The *physical signs* are usually well defined, and the area of hepatic dulness is uniformly increased, extending in some instances as low as the umbilicus. The enlargement, however, is not so great as in amyloid disease.

**Differential Diagnosis.**—Fatty infiltration of the liver is not apt to be mistaken for any other affection of this organ. The occurrence of general obesity, together with an entire absence of symptoms of obstruction to the portal vessels or bile-ducts or of other evidences of *fatty degeneration* (particularly feeble heart-sounds), will help to distinguish it from this latter condition. The etiologic factors above mentioned will also aid in the differentiation.



**Prognosis.**—This is decidedly favorable, as the function of the liver in many instances is not impaired in the slightest degree.

**Treatment.**—As the disease is of gradual development and long duration, a modification of the *diet* constitutes the first essential of the treatment. That prescribed under the Treatment of Amyloid Liver is admirably suited to this affection. Saccharine and farinaceous articles of food (potatoes, oatmeal, and sweetmeats) must be eschewed. Wheat-bread must be partaken of sparingly, and in its place gluten- and bran-bread or crusts of French rolls should be used. Fish, lean meats, fresh vegetables, and fruits are also allowable. Alcoholic beverages must be interdicted.

Graduated daily exercise and Turkish or Russian baths, judiciously used, are important factors in the treatment. Medicinally, the salts of the alkalis are highly recommended: sodium sulphate (in dram—4.0—doses, taken on an empty stomach) and ammonium carbonate (gr. xv to xxx—1.0 to 2.0—in twenty-four hours).

## FATTY DEGENERATION OF THE LIVER.

**Definition.**—A conversion of the albuminates of the cells into fat; it is characterized anatomically by a destruction of the liver-substance, with atrophy of the organ, and clinically by biliary, gastro-intestinal, cardiac, and renal symptoms.

**Pathology.**—On examining a liver that is the seat of marked fatty degeneration the organ is found smaller than normally, and the substance is light yellow in color, soft, pliable, and easily torn. On section the relation between the interlobular connective tissue and the acini is lost, the latter being replaced by fat-cells and oil-droplets. Scattered areas of pigmentation may be observed throughout the organ.

*Microscopically*, the cells lose their shape and become globular; the nuclei tend to coalesce, and finally disappear, together with the cell-wall, giving rise to compound globule-cells, which do not tend to coalesce and are stained black by osmic acid. Crystals, granular debris, Lener's spheres, cholesterin, tyrosin, and phosphatic crystals are also found in this form of granular change.

**Etiology.**—The following are among the recognized causes of the affection: (a) The excessive use of beer or alcoholic liquors. (b) It may be a sequence of amyloid disease, and hence result from any of the causes of the latter. (c) Diminution of the oxygen-supply to the tissues, occurring in phosphorus-, chloroform-, or arsenic-poisoning and in certain wasting diseases (carcinoma, phthisis, and chronic dysentery). (d) It may occur as a complication in the grave anemias, especially pernicious anemia, and in acute infectious diseases and the intoxications; also as a part of the pathology of acute yellow atrophy of the liver.

**Symptoms.**—I feel convinced that partial or *mild cases* of fatty degeneration of the liver present no morbid symptoms of diagnostic import. Pain, jaundice, and ascites may occur separately or conjointly, but form the exception rather than the rule. The *severe forms* are characterized

by the symptoms seen in phosphorus-poisoning and acute yellow atrophy, to the discussion of which the reader is referred.

**Complications.**—The disease may be complicated with fatty change in the *kidneys*. Under these circumstances the *urine* is diminished in amount, of low specific gravity, and contains an abundance of albumin, fatty or oily casts, and crystals of cholesterin, leucin, and tyrosin. In marked cases there is a very *feeble and irregular cardiac impulse*, accompanied by attacks of *vertigo* and *syncope*, the latter symptoms indicating beginning degeneration of the cardiac muscle. *Edema* of the lower extremities and *anasarca* may occur as complications of this condition.

The **physical signs** elicited by *palpation* and *percussion* show increasing diminution in the size of the liver as the disease advances.

**Diagnosis.**—The chief diagnostic points of fatty degeneration may be summated thus: (a) A history of alcoholism, of poisoning by drugs (arsenic, phosphorus, or chloroform), or of an acute infectious disease (acute yellow atrophy); (b) Grave general symptoms, as albuminuria, edema, ascites, cardiac failure, terminating often in acholia or cholemia; (c) Progressive diminution in the size of the organ. When these occur conjointly the diagnosis is established beyond a doubt.

**Prognosis.**—The prognosis is entirely dependent upon the cause. If due to an excessive use of stimulants, the process, if recognized early, may be arrested; if associated with acute yellow atrophy or other infectious disease, the outlook is unpromising.

**Treatment.**—The indications for treatment may be divided into the *dietetic*, *hygienic*, and *medicinal*. The same precautions regarding diet should be observed as in fatty infiltration. An open-air existence, short of injurious exposure, aided by hot salt-water, Turkish, or Russian baths, under restriction, is sure to improve the general condition of the patient.

The medicinal treatment varies according to the cause of the disease. If due to grave anemia, iron (tinct. ferri chlorid. or syrup. ferri. iodid.) may be given in ascending doses. Poisoning by drugs that produce fatty degeneration of the liver is to be combated by their respective antidotes. Gastro-intestinal disturbances, if coexistent, demand appropriate treatment. For the latter Frerichs recommends highly the salts of the alkalis (sodium sulphate in dram—4.0—doses taken on an empty stomach and ammonium carbonate). Ascites and cardiac asthenia, when occurring as complications, must be met by suitable measures.

## PERIHEPATITIS.

### ACUTE PERIHEPATITIS.

(*Pyo-pneumothorax Subphrenicus*.)

**Definition.**—An inflammation, either suppurative or fibrinous, of the peritoneal covering of the liver and the corresponding portion of the diaphragm.

**Pathology.**—The morbid changes may consist in a purely plastic inflammation, the serous layers being thickened, opaque, and covered with a fibrinous exudate leading to adhesion. In the majority of cases,

however, the inflammatory product is chiefly purulent, and is ribboned by fibrous bands so as to form circumscribed areas, filled with pus, lying between the liver and the diaphragm; this constitutes the *subphrenic abscess*. The latter is found more commonly to the right than to the left of the suspensory ligament. It may contain much pus (1 quart—1 liter—or even more), which in most cases is mixed with air or gas derived from the gastro-intestinal canal. Rarely, bilirubin-crystals are found, betraying the presence of bile. If the latter be present in large amount, the pus assumes an ochre-yellow hue.

**Etiology.**—The *fibrinous variety* may result from the direct extension of one or other of the acute forms of inflammation of the liver (abscess, hydatid cyst), from a pleurisy spreading along the lymphatics in the diaphragm, or from traumatism—particularly a blow. The *suppurative form* (*pyo-pneumothorax subphrenicus*, Leyden) may be caused in the same manner as the former, but far oftener—in more than one-half of the instances—it follows perforation of a gastric ulcer, and far less commonly perforation of a duodenal or colonic ulcer. Appendicitis and penetrating wounds are not infrequent causes. Perihepatitis is a grave complicating event in carcinoma (of the stomach, esophagus, and intestines), in lobar pneumonia, and purulent pleuritis.

**Symptoms.**—Those of the *acute fibrinous variety* are either altogether missing or too vague to admit of correct interpretation. The coappearance, however, of severe *pain*, increased on deep breathing, and *tenderness* over a circumscribed area either in the right hypochondrium or the epigastrium, after the action of some known cause or the occurrence of one of the causative affections, is strongly suggestive of this form of the complaint. A *friction-sound* may at times be heard below the seventh rib in the mammillary and the ninth rib in the axillary line, or over the epigastrium, as in two cases in my practice. It is of short duration, and is limited usually to the end of inspiration. It must be recollected that plastic pleurisy may be an associated condition.

In *suppurative perihepatitis* the symptoms are sometimes screened by those characterizing the special causative complaint; but in my experience, in cases due to perforation—the most common cause—the *onset* is rapid and severe, and is marked by *acute pain* referred to a circumscribed spot in the hepatic region, great *tenderness*, rapid, embarrassed, and *painful respiration* (owing to implication of the diaphragm), by *vomiting* (often bilious, though at times hemorrhagic) or *nausea*, and by faint *jaundice* in some cases. Shortly the *general features* of circumscribed peritoneal abscess also appear—rigors, irregular fever, sweats, and progressive prostration and emaciation.

**Physical Signs.**—*Inspection* discloses bulging of the right hypochondrium and often of the epigastrium. The same regions are immobile, but this is best appreciated by *palpation*. The anterior edge of the liver is felt even as low as the umbilical level. *Percussion* reveals a variable increase of hepatic dulness upward, sometimes touching the fourth rib. The upper level of the fluid is movable on changing the position of the patient, and this is particularly striking if air or gas is contained in the abscess; the presence of the latter also causes a zone of tympanitic resonance above the dull area, while overlying the latter



there is the semi-tympanitic area of the retracted lung. *Auscultation* reveals an absence of breath-sounds and of the vocal resonance over the dull and tympanitic areas, while the respiratory sounds over the displaced lung are broncho-vesicular.

**Differential Diagnosis.**—Acute perihepatitis often remains unrecognized during life. It may be confounded with *empyema* of the right side, but the two conditions have different modes of development. Perihepatitis is preceded and accompanied by abdominal symptoms; empyema manifests thoracic symptoms—*e. g.* cough and pleuritic pain. At a later stage the exaggerated respiratory murmur above the dull area, the slighter cardiac displacement toward the left, and the greater hepatic displacement downward in suppurative perihepatitis aid in the differentiation. The introduction of the trocar in the seventh or eighth intercostal space in the mid-axillary line may also be helpful, especially if the exudate be found to contain bile-pigment. *Pfuhl's sign*—the more ready escape of the fluid during inspiration on aspiration of abscesses below the diaphragm—may not be without value. The points narrated above may likewise serve to separate pyo-pneumothorax from suppurative perihepatitis (see also Pneumothorax, p. 568).

**Course and Prognosis.**—In the milder or fibrinous variety the outlook is favorable and the course is brief. On the other hand, the suppurative type due to perforation, if not early brought under proper surgical treatment, often terminates unfavorably by gradual asthenia. Rarely the pus is resorbed, or it may find an outlet through the lungs, abdominal walls, or other avenue, followed by slow recovery.

The **treatment** is the same as for localized peritonitis. The first evidence of the presence of pus is the signal for appropriate surgical interference—evacuation and drainage.

#### CHRONIC PERIHEPATITIS.

(*Zuckergussleber.*)

This affection is a chronic inflammation of the perihepatic fibrous membrane, which becomes opaque and thickened. Contraction of this capsule ensues, with compression of the liver and atrophy to even one-half the size of the normal organ (as in a case reported by Rumpf<sup>1</sup>), and partial or total occlusion of the vessel and bile-ducts. Perhaps these changes are most marked in cases that follow acute suppurative perihepatitis. Genuine instances show no hyperplasia of the interstitial connective tissue; hence the condition is closely related pathologically to “Glissonian cirrhosis” (*vide* p. 886).

The main **causes** of chronic perihepatitis are great and protracted local pressure, as from a corset, and certain occupations. It may represent a portion of a more general chronic inflammation of the serosæ. Finally, I am of the belief that syphilis is the leading single cause, and could discover no other factor present in two cases that yielded to anti-syphilitic treatment.

The **diagnosis** is generally problematic. Of especial clinical worth are the etiology, pain in the right hypochondriac region—particularly in cases due to syphilis—absence of the signs of stasis of the gastrointestinal tract, and the very protracted course.

<sup>1</sup> *Deutsch. Arch. f. klin. Med.*, March 13, 1895.

The **treatment** is purely palliative, apart from the effort to remove the special cause, whether this be syphilis, occupation, or other influential factor.

## ABSCESS OF THE LIVER.

(*Hepatic Abscess; Suppurative Hepatitis.*)

**Definition.**—A circumscribed collection of pus in the hepatic parenchyma.

**Pathology.**—If examined *in situ*, a liver that is the seat of abscess-formation is usually found to be symmetrically enlarged, and on careful palpation one or more areas of fluctuation (either deep or superficial, according to the location of the abscess) may be detected. If single, its position is usually in the right lobe near the convexity of the organ (70 per cent. of cases). The tissue surrounding the abscess-wall is usually deeply injected, the wall itself in acute cases being poorly defined, but grayish in color, irregular and shreddy, and composed of necrotic liver-cells, pus-corpuscles, and often amebæ. In chronic cases it becomes greatly thickened and often cartilaginous in appearance.

Microscopically, the hepatic cells are altered in shape and devoid of nuclei; they undergo rapid degeneration. A round-celled infiltration occurs about the blood-vessels, their walls being filled with small emboli containing innumerable staphylococci and streptococci. As the suppurative process continues liquefaction-necrosis occurs, resulting in complete destruction of the hepatic parenchyma.

The amount of fluid contained in a liver-abscess may exceed 2 or 3 quarts (2–3 liters), and its color varies from grayish-white to a creamy, reddish-brown. The collection in some instances resembles healthy pus. I have spoken of the methods of infection and of some of the different varieties of hepatic abscess in the discussion of Dysentery (see p. 96).

Various odors are described, depending largely on the extent of bacterial invasion and the degree of necrosis. In this connection it may be said that in amebic dysentery, with abscess of the liver as a complication, the abscess is often single (involving more often the right lobe), whereas other forms due to septic infection give rise to multiple abscesses.

In the latter instances the surface of the organ presents many small yellow areas beneath the capsule, varying from 5 to 15 mm. ( $\frac{1}{8}$ – $\frac{3}{8}$  inches) in diameter. Usually, in such cases infection has taken place through the portal circulation, and on section the appearances of a suppurative pylephlebitis present themselves. If thrombi have formed in the portal tributaries, localized necrotic areas are the result, but more often the invasion affects the whole portal system, the liver being riddled with abscesses. If the abscess is secondary to obstruction by gall-stones or inspissated bile, the ducts are greatly distended and the gall-bladder is filled with pus mixed with bile.

**Etiology.**—Idiopathic abscess of the liver is rare even in tropical climates. The affection, even when excited by mechanical causes, as traumatism or obstruction by gall-stones, is invariably septic in cha-

racter, and the infecting material reaches the interior through the hepatic vessels or the biliary passages.

*Septic emboli* enter the liver by means of the vascular system through the hepatic artery or portal vein, the latter being the more common channel of transmission. Gastric ulcers, or the ulceration occurring in dysentery, typhoid fever, typhilitis (?), or appendicitis, may be followed by a purulent portal pyelphlebitis, resulting in abscess-formation. In general pyemic processes or in bone-suppurations of long standing the germs enter the venous circulation, traverse the intralobular pulmonary plexuses, and enter the liver through the hepatic artery. Suppurating wounds of the head are not uncommonly followed by abscess of the liver. Because of obstruction of the common duct by gall-stones, either from pressure-necrosis or owing to the decomposition of accumulated bile, pathogenic organisms may enter the liver and cause abscess-formation through the medium of the bile-ducts. The most common method of infection, however, is *through the portal vein*. Among other causes may be mentioned *foreign bodies* travelling up the ducts, as parasites, round-worms, liver-flukes; also, more rarely, suppuero-perforation by mechanical irritants (needles, pins, fish-bones, and the like), and supuration occurring in the course of an hydatid cyst. Leick has tabulated 19 cases of hepatic abscess caused by the *ascaris lumbricoides*.

**Symptoms.**—In a typical case of hepatic abscess the most prominent symptoms are—*hectic temperature, pain, tenderness, and enlargement of the organ*, and often *slight jaundice*, although it must not be forgotten that any or all of these may be absent during the development of an abscess. The multiple abscesses occurring in pyemic conditions, which are frequently diagnosed when in view upon the postmortem table, form an instance of this.

To facilitate the subject I shall consider the more important symptoms *seriatim*: *Pain* is circumscribed to the hepatic region, and radiates to the right shoulder in conjunction with the other symptoms and physical signs; it is very characteristic, although not pathognomonic of hepatic abscess. In the earlier stages this symptom is not pronounced unless the abscess or abscesses lie superficially. It is usually of a dull, boring character, differing in severity with the patient's position; it is usually aggravated by pressure over the costal margin and by lying on the left side, this tending to drag the liver by its own weight from its normal position. Luschka explains the radiation of pain to the right shoulder by stating that filaments of the phrenic nerves that distribute themselves in the suspensory ligament and Glisson's capsule are irritated. The phrenic arises from the third, fourth, and fifth cervical nerves, and, as the fourth supplies sensation to the right shoulder, the impression is thus transmitted through the central nervous system.

In acute cases accompanied by rapid destruction of the hepatic tissues the *temperature* usually rises rapidly, reaching 103° or 104° F. (39.4°–40° C.) in the course of from twenty-four to thirty-six hours. Its course, however, is irregular and intermittent, and it may be hectic in character; just as often it resembles a tertian or quartan intermittent or a remittent temperature. *Rigors* or *decided chills* frequently accompany the rise of temperature, and during the decline profuse sweatings may take place, thus simulating to a certain extent the symptoms of malarial



fever. In chronic abscess of the liver pyrexia may be entirely absent. Less commonly the temperature may remain continuously high, with slight morning and evening exacerbations and remissions. The *pulse* is usually rapid in proportion to the temperature.

The **physical signs** in a case of hepatic abscess are always present to a greater or less degree, and are often pathognomonic.

*Inspection* may reveal nothing during the entire course of the disease, although in cases accompanied by intense congestion in which the abscess involves the anterior surface of the right lobe, bulging of the ribs on that side will occur, with a marked prominence in the hypochondriac region extending three or more finger-breadths below the costal margin.

*Palpation* confirms inspection and reveals tenderness on pressure below the costal margin in the mammary line. The liver, if projecting below the edge of the ribs, is usually enlarged uniformly, unless the abscess involves the surface of the margin. As the upper right lobe is more often involved, especially in a large single abscess, the increase in size is in an upward direction, thus rendering palpation negative. In rare instances the abscess gives rise to fluctuation on palpation, and if the peritoneum be inflamed a friction fremitus may be detected.

*Percussion*.—The area of hepatic dulness may be increased uniformly, but it is usually most marked upward and to the right, in some instances reaching at the anterior axillary line to the fifth rib, and posteriorly to the level of the angle of the scapula. This high position of the upper boundary of dulness which starts about the nipple-line serves to differentiate abscess from other affections of the liver, in which the enlargement extends in a downward direction.

**Other Symptoms**.—The *skin* is pale and shows slight icterus, the *conjunctivæ* being often bile-stained; intense *jaundice*, however, is rare. Progressive loss of flesh and strength, with *gastro-intestinal disturbance* (fulness in the epigastrium, flatulence, water-brash, nausea, and occasional vomiting), are common symptoms at the onset. The *bowels* are variable, and constipation usually alternates with diarrhea, the stools in some cases containing the ameba coli. *Ascites* may develop from pressure on the inferior vena cavæ, but these cases are rare. The *spleen* may undergo active hyperplasia in acute abscess-formation. *Pulmonary symptoms* (severe cough, characteristic reddish-brown sputum, resembling anchovy sauce, broncho-vesicular breathing, râles) are commonly present; they are due to compression of the base of the lung by the abscess pressing upon the diaphragm. In fatal cases certain *nervous symptoms* (muttering delirium, cephalalgia, subsultus tendinum, stupor, coma) make their appearance.

**Complications and Sequelæ**.—The abscess may perforate into the pleural cavity (pyothorax), bronchi, lungs, intestinal tract, stomach, pericardium, peritoneal cavity, or externally through the abdominal wall, giving rise to various symptoms. If rupture occurs into the intestinal tract, sudden diarrhea, with the discharge of large quantities of pus, takes place; there is then an amelioration of the pain, fever, and other symptoms. If the rupture is into the lung, the physical signs will reveal the sudden development of weak, tubular breathing over the base, with increased tactile fremitus and percussion-dulness, together

with the occurrence of profuse and typical expectoration. Reese and Lafleur found the ameba coli in the bronchial discharge. S. Flexner has reported two cases of amebic abscess of the liver in which perforation into the vena cava occurred. Rupture into the abdominal cavity gives rise to the rapid development of a purulent peritonitis that is often fatal. Rarely, the abscess is emptied into the pericardium, giving rise to fatal acute pericarditis. Septic emboli have been known to lodge in the circle of Willis, producing fatal brain-abscess.

**Diagnosis.**—The clinical symptoms of hepatic abscess are of diagnostic importance only when taken in the aggregate, since the pain, fever, enlargement, and even hectic symptoms occur singly in other conditions unaccompanied by suppuration. The principal points in the establishment of the diagnosis of the affection may be summed up as follows: Residence in tropical countries, the previous existence of typhoid or dysenteric ulceration (or other gastro-intestinal inflammation), the characteristic expectoration, enlargement of the liver, with pain and tenderness on pressure, and in some instances fluctuation on palpation. Lastly, aspiration may reveal pus-corpuscles, hepatic cells, staphylococci and streptococci, the ameba, and bile-pigment, which when found are pathognomonic; if the abscess be secondary to an echinococcus cyst, the presence of hooklets will be detected.

**Differential Diagnosis.**—Hepatic abscess may be misdiagnosed for *empyema*, *malarial fever*, and *hepatic calculi*.

**Empyema.**—The mode of onset and the physical signs peculiar to this condition, if studied carefully, are entirely different from those of abscess. In empyema there may be the history of a perforating wound of the chest, the rupture of a bronchiectatic or tuberculous cavity, or the pre-existence of a sero-fibrinous pleurisy; whereas hepatic abscess may be preceded by an attack of amebic dysentery or intestinal ulceration, or it may follow the impaction of biliary calculi. In both there may be the occurrence of a hectic temperature, with chills and sweating; but in empyema cough and dyspnea are prominent, and, if the pleural cavity communicates with a bronchus, profuse muco-purulent expectoration containing pus-cells, staphylococci, streptococci, and in many cases elastic tissue and tubercle bacilli. Rarely, an abscess of the liver penetrates the diaphragm, and, entering the bronchi, is expectorated. The recognition of hepatic abscess under these circumstances is to be based mainly upon clear evidence of the affection prior to the occurrence of perforation, and copious, blood-tinted, purulent expectoration. The detection of the ameba coli in the sputum alone would set the diagnosis at rest. The contents of hepatic abscess obtained by aspiration consist of the micro-organisms of suppuration, and in addition broken-down liver-cells, bile-pigment, and in some cases the ameba coli. Inspection in empyema reveals bulging of the intercostal spaces on that side, while percussion gives absolute flatness over the base of the chest, rising posteriorly and changing with the change from a dorsal to a sitting position. Above the area of flatness we find either a normal pulmonary note or hyper-resonance. In abscess of the liver the lung is slightly displaced upward, being often bound to the diaphragm by adhesions; and the upper boundary of dulness is lower, particularly in front, and is not changed with the decubitus of the patient.

## HEPATIC ABSCESS.

History of traumatism, dysentery, intestinal ulceration, or residence in tropical countries.

Icteric character of the temperature—high every evening and low every morning; irregular chills, followed by fevers and sweatings.

An irregular, fluctuating tumor or multiple nodules in the liver; no splenic enlargement; rapid emaciation, with or without jaundice, but no cachexia.

Blood shows simple anemia and leukocytosis, and in marked cases disintegration of red blood-cells.

Abscess-contents show the staphylococci, streptococci, amebæ, or bacillus coli communis, and pus.

## MALARIA.

History of previous attacks. Residence in warm, damp climates among the lowlands.

Regularly recurrent rise of the temperature (intermittent or remittent, quotidian, tertian, quartan, or septinarian), and the rise occurring during the chill, followed by profuse sweating; chills more often in morning.

The spleen is enlarged; also there is a yellow-brown coloration of the skin, more or less marked; and, in long-standing cases, the occurrence of *cachexia*.

The presence of the hematozoa of Laveran and free pigment in the blood.

Absent.

*Impacted Calculi.*—In this condition attacks of *hepatic colic* are often first noticed, followed by jaundice, and, if impaction be not absolute, by the occurrence of stones in the feces. In abscess the pain is not paroxysmal, but dull and boring in character, increasing in severity as the disease progresses. In *chronic impaction* jaundice, dull pain over the hepatic area, distention of the gall-bladder (which in some instances may be palpated), and clay-colored feces, constitute the principal symptoms. There occurs also an intermittent fever as in hepatic abscess, but it is occasional—*i. e.* the febrile paroxysms recur at longer intervals. Again, the course of intermittent hepatic fever associated with biliary calculi is much more chronic than the fever-stage of suppurative hepatitis. On the other hand, in abscess of the liver jaundice is comparatively rare, and, unless the abscess rupture into the gastro-intestinal tract, the stools show nothing abnormal. In some instances biliary abscesses may follow impacted calculi, and it is always a secondary affection.

Among other liver-conditions that are liable to be mistaken for hepatic abscess may be mentioned *carcinoma*, *hypertrophic cirrhosis*, and *hydatid cyst*, the differential diagnosis of which will be spoken of under these diseases.

**Prognosis.**—The prognosis of hepatic abscess is unfavorable, the disease generally progressing to a rapidly fatal termination. Prompt evacuation of the abscess when its location can be detected, however, may be successfully performed. The mortality ranges from 50 to 60 per cent. In rare cases the walls of the abscess become calcified and the disease remains latent. The single large abscess that most often follows dysentery offers the best opportunity for surgical measures.

**Treatment.**—Barring operation, the treatment of abscess of the liver is purely symptomatic, being in many instances identical with that of septico-pyemia. The temperature often responds to repeated spongings with cool water (65° F—18.3° C.). For the pain mustard-poultices, the turpentine stupe, or hot fomentations over the hepatic area, in conjunction with full internal doses of opium, prove beneficial. Full and free stimulation and the free exhibition of quinin as soon as the



condition is detected both support the system and control, in a measure, the pyemic process. If the abscess be single and localized, prompt evacuation should be resorted to.

## ACUTE YELLOW ATROPHY.

(*Malignant Jaundice; Icterus Gravis.*)

**Definition.**—An acute and probably infectious disease, characterized by a rapid destruction of the parenchyma of the liver and by a diminution in the size of the organ; also by jaundice, hemorrhage, and grave cerebral phenomena.

**Pathology.**—Macroscopically, in a case of acute yellow atrophy the liver is seen to be much reduced in size, weighing but 15 or 20 ounces (480.0–640.0), instead of its normal weight (50 oz.—1.6 kgms.). The capsule is shrivelled and the organ is of a pulpy consistence, and changed in appearance from a mahogany-brown to a grayish-yellow hue. Sometimes the liver is primarily enlarged. The cut section often presents areas of red and yellow discoloration, the so-called “red atrophy” and “yellow atrophy,” the former being a later stage of the latter. The red appearance is due to an excess of blood in the capillaries, with free pigment that has been liberated by destruction of the red blood-cells. Microscopic examination reveals destruction or necrosis of the hepatic cells. The nuclei have disappeared, and the cell-wall contains a number of fat-globules of various sizes containing free pigment. In advanced cases, accompanied by total disintegration of the cells, fat-droplets, granular debris, cholesterin-plates, leucin-spheres, tyrosin-needles (first discovered by Frerichs, both in the cells and in the blood-vessels), and crystals of bilirubin may be found. The common duct is patulous, and the gall-bladder is usually empty.

In well-marked cases both the *heart* and *kidneys* show evidences of fatty degeneration, the kidneys often showing ecchymotic areas. The *spleen* is greatly enlarged from active congestion, giving rise to the so-called “acute splenic tumor.” The splenic substance is soft and easily torn, and on section the organ often drips blood. The *skin* and *mucous membranes* may be the seat of numerous ecchymoses, and dropsy of the pericardial and other serous cavities is frequently noted. The *blood* is dark and fluid, and under the microscope is seen to contain disintegrated red corpuscles, with crystals of leucin and tyrosin.

**Etiology.**—The causes of acute yellow atrophy are both primary and secondary. *Primary* or idiopathic acute yellow atrophy is rare and its course as yet unsettled. Among the *secondary* predisposing causes may be mentioned age (fifteen to thirty-five years), female sex, parturition, syphilis, and certain acute fevers (puerperal fever, typhoid, septicemia, malaria). Acute phosphorus-poisoning sometimes presents changes resembling those of acute yellow atrophy. The disease rarely accompanies cirrhosis of the liver, and may follow a debauch. Rarely, an endemic form is assumed, but the exciting cause is thus far unknown. The disease is probably microörganismal or toxic in nature, and

although various germs have been discovered, their claim to specificity has not been established.

**Symptoms.**—The clinical history of acute yellow atrophy varies considerably in the early stages of the disease, the graver symptoms of the later stage alone being pathognomonic. The attack is usually ushered in by *headache, malaise, anorexia, nausea, and vomiting, moderate fever*, and after a few days *jaundice* appears. *Physical examination* at this time shows the area of hepatic dulness to be normal or only slightly increased. After a period varying from a few days to two or three weeks (during which the typical features of catarrhal jaundice have been present), grave *nervous and cerebral* symptoms present themselves, as restlessness and violent headache, followed by delirium, which often becomes maniacal. *Convulsions* then appear, and are succeeded by stupor and coma, the latter occurring usually within forty-eight hours from the onset of the period of cerebral excitement. Often *coarse tremors* are noticed in the voluntary muscles, and with the onset of the second stage the jaundice usually deepens.

The *temperature* often remains normal until just before death, when it may rise one or two degrees. The *pulse* is much diminished both in volume and tension, and is rapid in proportion to the temperature. The *tongue* at the onset is covered with a light coating, most marked on the dorsum and tip. Later, it changes to a thick yellow color and becomes dry and fissured, with the development of a typhoid state. Vomiting appears usually during the premonitory stage and often becomes intense; the *vomit* consists at first simply of the gastric contents, which later in the disease becomes mixed with blood (hematemesis). *Hemorrhages* also frequently occur into the skin (ecchymoses) and from the mucous membranes, giving rise to epistaxis, hematuria, melena, hemoptysis, and menorrhagia. *Constipation* with clay-colored stools is common.

The *urine* in acute yellow atrophy is often scanty in amount, high colored, and shows an increase in specific gravity (1028–1032). The *urea* is greatly diminished, but bile-pigments and albumin, tube-casts, *leucin* and *tyrosin* are found both on chemical and microscopic examination. The latter can be easily demonstrated by allowing a drop of the urine to evaporate on a cover-glass and examining under the microscope. Tyrosin-crystals are deposited in the form of sheaves and rosettes, leucin as globular masses. These bodies are not constantly present. Thus, out of 34 cases collected by Thierfelder, in which the urine was examined in this relation, “in 7 the result was negative; in 17 both were found; in 3 tyrosin only; in 7 leucin only.” Among other products found in the urine worthy of mention are creatinin, lactic and sarcolactic acids, and other bodies belonging to the fatty acid series.

Acute yellow atrophy of the liver is a striking example of acid-intoxication due to rapid and widespread destruction of proteids as the source of the fatty acids—sarcolactic, lactic, diacetic, and  $\beta$ -oxybutyric. It is probable that the rare nervous phenomena of the disease are, in part at least, due to the diminished alkalinity of the blood arising from the abnormal metabolism.

The **physical signs** reveal tenderness over the hepatic region, often amounting to actual pain. During the second stage, in extreme cases, the

edges of the organ cannot be palpated under the costal margin. Percussion, moreover, shows a great diminution in the size of the liver, the area of dulness in a case recorded by Harley extending over but 1 inch (2.5 cm.) in the mammary line and  $1\frac{1}{4}$  inches (3.1 cm.), measured perpendicularly, in the mid-axillary line.

The left lobe is often the first to show physical signs of atrophy, percussion giving tympany instead of flatness in the upper epigastric region. As the atrophy continues the tympany extends below the seventh rib from above and advances upward from the costal margin, leaving but a small circumscribed area of hepatic dulness. The atrophy is usually progressive until death occurs, although favorable cases have been recorded in which the liver increased in size perceptibly during recovery (Harley, p. 260).

**Diagnosis.**—The symptoms occurring during the second stage of the disease are usually so characteristic as to leave little doubt concerning the diagnosis. The occurrence of gradually increasing jaundice with vomiting, grave delirium, hemorrhages, the presence of an immense amount of bile, with leucin and tyrosin, in the urine, and greatly diminished size of the liver, all combine to form a typical clinical picture. Unfortunately, leucin and tyrosin are also found in the urine in acute phosphorus-poisoning and rarely in severe acute infective diseases.

**Differential Diagnosis.**—In *hypertrophic cirrhosis* the onset is more gradual. There is generally a negative previous history; and examination of the urine fails to reveal leucin and tyrosin; fever is rarely present in cirrhosis, and the physical signs often show a considerable increase in the area of hepatic dulness.

The differential diagnosis between this disease and phosphorus-poisoning is given under the latter condition (*vide infra*).

The **prognosis** is almost invariably fatal, since every case of true yellow atrophy is associated with a destruction of liver-cells that is accompanied by acute toxemia.

**Treatment.**—As yet no specific treatment has been discovered, all remedies used being directed to the relief of symptomatic indications. The gastro-intestinal system should be relieved at the onset by divided doses of calomel. For the vomiting cracked ice, with 1-minim (0.066) doses of the wine of ipecac repeated every half hour or divided doses of opium, may be given. Marked nervous phenomena with delirium I have seen controlled by cool baths and the ice-cap, together with camphor, chloral, or other antispasmodics used internally. Free stimulation should be begun early and persisted in throughout the course of the disease.

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## THE LIVER IN PHOSPHORUS-POISONING.

FOLLOWING the ingestion of a dose of phosphorus varying from gr.  $\frac{1}{8}$  to gr. 1 (0.008–0.0648) symptoms of poisoning manifest themselves (Taylor, Wormley) as follows:

After a period of time varying from three to twelve hours a sense of wretchedness, nausea, abdominal pain (not intense), and often vomiting,



occur. The *vomit* consists of the gastric contents, with bile, and during the first few hours it may contain phosphorus, which gives it a luminous appearance in the dark.

After the second or third day the vomiting usually ceases with the appearance of *jaundice*, which may become intense as the process continues. Later in the course of the case emesis recommences, the vomita consisting of altered blood, giving rise to the so-called "black vomit." At this stage *nervous symptoms* usually manifest themselves (headache, insomnia, vertigo, and delirium, with convulsions and coma in fatal cases), death closing the scene usually in from thirty-six to forty-eight hours.

The *bowels* are constipated, although attacks of diarrhea may supervene, the evacuations being in some instances phosphorescent.

*Fever* is irregular and usually is not marked, the temperature swinging from 99° to 101° F. (37.2°–38.3° C.). In fatal cases the temperature may become subnormal just before death.

The *urine* is scanty, of high specific gravity, and contains bile, bile-acids, albumin, sarcocollin, and in rare cases leucin and tyrosin (Wood). Renal epithelium and free fat-globules have also been found. When occurring in pregnant women, abortion or miscarriage invariably follows.

*Physical examination* reveals a liver uniformly enlarged and tender on pressure. In protracted cases atrophy of the organ may rarely occur.

**Etiology.**—The most common causes are—(a) Occupation, workers in match-factories being the most frequent sufferers; (b) The accidental swallowing of phosphorus (*e. g.* rat-poison, friction-match heads).

**Pathology.**—On opening the abdominal cavity in a case of phosphorus-poisoning the liver is seen to extend below the costal margin, its surface being lighter in color than normal and mottled in appearance, and its substance softer in consistence and friable.

The cut section presents marked evidences of fatty degeneration, the acini being lighter in color than the interlobular tissue. Portions of the hepatic parenchyma are deeply bile-stained, and on scraping the cut surface bile- and fat-globules will be found on the edge of the knife. The gall-bladder may be either full or empty. Microscopically, disintegrated liver-cells, fat-globules, granular debris, biliary coloring-matter, leucin-spheres, cholesterin-plates, and tyrosin-needles are noted.

The *gastric mucosa* is found thickened, opaque, and yellow-white in appearance, due, as pointed out by Virchow, to a universal gastro-adenitis, and not to the local action of the poison. Ulcerative or erosive gastritis is very rare in phosphorus-poisoning.

The *kidneys* may show beginning atrophy, the epithelium in the cortices undergoing granular and fatty degeneration, with final destruction of the cells.

The *blood* is dark, fluid, and not easily coagulable. Concato found that during life the white corpuscles are increased in number, and that the red are changed in shape and smaller than normal (Wood). Petechiæ and ecchymoses frequently appear in all parts of the body.

**Diagnosis.**—The diagnosis of acute phosphorus-poisoning is always extremely difficult and often impossible. The disease with which it is

most apt to become confounded is *acute yellow atrophy of the liver*. The differential points may be summated as follows:

## ACUTE PHOSPHORUS-POISONING.

There is a history of accidental ingestion of poison (friction-match heads, rat-poison) or occupation.

The onset is sudden; violent nausea, vomiting, and pain over the region of the liver. Jaundice appears on the second or third day.

Nervous symptoms appear late in the disease—always preceded by jaundice.

The vomit and stools are phosphorescent. Black vomit precedes death.

Temporary arrest of symptoms between the occurrence of jaundice and black vomit.

Sarcolactic acid is present in the urine, and rarely leucin and tyrosin.

## ACUTE YELLOW ATROPHY.

There may be an endemic history.

A slow onset—malaise, slight fever, with nausea and vomiting; jaundice is a beginning symptom.

Nervous symptoms may appear early, even before the occurrence of jaundice.

Black vomit occurs early and persists throughout.

Progressive march of symptoms with no remission.

Leucin and tyrosin are common in the urine.

**Prognosis and Duration.**—The prognosis in phosphorus-poisoning is bad, as small a dose as gr.  $\frac{1}{8}$  (0.008) of white phosphorus having caused death (Wormley). The duration is usually from one to six days, although the symptoms have been known to persist for twelve days before death. In violent cases the end may come within twenty-four hours.

**Treatment.**—The initial plan of treatment is by causing emesis to free the system of the poison that still remains undigested. For this purpose copper sulphate (gr. x—0.648) in divided doses (gr. ij or iij—0.129 or 0.194—every five minutes) should be given until free vomiting occurs. As copper sulphate is a chemical antidote, forming with phosphorus black copper phosphid, it should be continued in less frequently repeated doses (gr. ij—0.129—every half hour) and guarded by morphia to prevent vomiting. If emetics by the mouth fail to afford relief, apomorphin muriate (gr.  $\frac{1}{5}$ —0.0129), hypodermically, may be resorted to. The free evacuation of the stomach should be followed by the administration of the French oil of turpentine. Wood recommends that 1 part be given to every 100th part of the poison ingested. Ordinary turpentine is useless, but combined with mucilage of acacia 2 fluidrams (8.0) of French oil of turpentine may be given every fifteen minutes until 1 ounce (32.0) has been taken.

Alkalies (magnesia) have been given, but are practically valueless. Free purgation should be effected if possible by Rochelle salts or magnesium citrate. Demulcent oils are never allowable, as they dissolve the phosphorus and hold it in solution. After absorption of the poison and degeneration of the tissues have taken place all known remedies are futile.

## CIRRHOSIS OF THE LIVER.

(*Sclerosis of the Liver; Nutmeg Liver; Gin-drinker's Liver; Interstitial Hepatitis.*)

**Definition.**—A chronic disease of the liver, characterized, pathologically, by an excess of connective tissue. It presents various biliary, gastro-intestinal, circulatory, and cerebral symptoms.

**Pathology.**—There are three pathologic varieties: (a) atrophic cirrhosis, or “gin-drinker’s liver”; (b) hypertrophic cirrhosis; and (c) biliary cirrhosis.

(a) **Atrophic Cirrhosis** (*Laennec’s*, or *alcoholic cirrhosis*) is the most common form, at least in the earliest stages, as Foxwell’s studies teach; the alcoholic (indurative) liver is more commonly enlarged than decreased in size. Morse<sup>1</sup> examined the records of 37 cases of cirrhosis, and found that among these there were 13 instances of enlarged liver, 11 of normal size, and 12 smaller than normal. In typical examples the capsule is thickened, the organ greatly reduced in size, hard, granular, and much altered in shape. On section (which resists the cutting-knife) the surface presents grayish-white bands of connective tissue surrounding yellowish areas (acini) that project above the surface from compression (hob-nails); hence the term “hob-nailed liver.”

*Microscopically*, the process is seen to commence as an increase in the connective-tissue element surrounding the terminal branches of the portal vein. Compression of the liver-cells and of the portal veins, with consequent obstruction of the circulation, constantly increases with the progress of the proliferation of the connective tissue and its secondary contraction. Atrophic changes in the hepatic cells, however, are often comparatively slight. The biliary canaliculi may be increased in number. Weigert and his disciples contend that atrophy or degeneration of the acini is often the primary change, and the connective-tissue production the secondary—filling the gap, so to speak.

In alcoholic cirrhosis the liver is sometimes large, smooth, or slightly granular, soft rather than hard, as ordinarily the case, and presents a light yellow color (*fatty cirrhosis*). Histologically, this is a form of true cirrhosis, as shown by the presence of an increase in the connective tissue, with which, however, fatty infiltration of the acini is associated.

(b) **Hypertrophic Cirrhosis** (Hanot).—On examining the liver *in situ* during hypertrophic cirrhosis the organ is found enlarged (sometimes enormously), the lower border projecting several fingers’ breadths below the ribs. The margin of the organ is well defined, the substance firmer than normal, and it cuts with difficulty. The organ is lighter in color than in health, and presents a yellow or mottled-green appearance. On treating a section with compound iodine solution (Lugol’s) the color changes to that of a deep mahogany-red. The acini are darker in hue than the interstitial tissue.

*Microscopically*, the peripheral zones of the acini are first seen to be the seat of a round-cell infiltration, with the formation of embryonal tissue; later, the interlobular connective tissue undergoes hyperplasia, causing obstruction of the biliary ducts with retention of bile, and subsequent atrophy of the liver-cells. New-formed bile-ducts are proliferated.

(c) **Biliary Cirrhosis.**—French writers have described “biliary cirrhosis” as opposed to a “portal cirrhosis” or atrophic. It results from obstruction of the bile-ducts; this causes retention of bile with swelling of the organ as a consequence. The irritant substances that are the result of stagnation of the bile start a cirrhotic process around the small bile-ducts (reactive inflammation). The *microscopic appearances* of the organ simulate those of hypertrophic cirrhosis; but the hepatic

<sup>1</sup> *Boston Med. and Surg. Journ.*, March 10, 1898.



cells are more deeply bile-stained. *Microscopically*, the first discoverable changes are spots of insular necrosis in the peripheral zones of the acini (Stengel). These are shortly replaced by proliferation of the interlobular connective tissues. The formation of new-ducts and liver-cells is common.

There is also a so-called Glissonian cirrhosis (perihepatitis) in which the capsule of the organ is surrounded by a dense white fibrinous membrane, which contracts, reducing the size of the liver and altering its shape. This I have described elsewhere (*vide* Chronic Hepatitis, p. 877). Syphilitic cirrhosis of the organ receives special consideration in the section devoted to Syphilis (*vide* p. 334).

**Etiology.**—(a) **Atrophic Cirrhosis.**—1. *Alcoholism.*—Freyhan, Osler, and I myself have found this causal factor operative in nearly all cases. Clinical history tends to prove that the stronger the alcoholic beverage (*e. g.* raw spirits) and the larger the amount consumed the sooner cirrhosis develops, although the quantity necessary to produce the disease varies greatly in different individuals. Doubtless by the side of alcoholism all other causes combined are comparatively insignificant.

2. *Spicy foods* are, according to some, classed as predisposing agents. Tiraboschi records a case that had long been induced by the use of spicy foods and by over-eating. In many cases ptomaines, the products of mal-assimilation through faulty digestion, are supposed to be the exciting cause.

3. *Male Sex and Middle Life.*—The cases produced by alcohol occur chiefly in males. According to my experience, females who misuse potable alcohols, particularly the more concentrated liquors, are less susceptible to the poison than males. Two-thirds of the fatal cases occur between the ages of 35 and 50 (Hawkins), although cases have been known to occur at both extremes of life. In children the disease is mostly caused by inherited syphilis, in which the organ is the seat of a general fibroid process. It may follow the acute infectious diseases, notably scarlet fever, in the young.

4. Certain *chronic diseases* (syphilis, rickets, diabetes, gout, malaria, carcinoma, tuberculosis) that favor the formation of connective tissue are apt to be complicated by cirrhosis, usually partial.

5. *Passive congestion*, secondary to chronic cardiac lesions or to obstructive lung-disease, not infrequently gives rise to hepatic cirrhosis.

6. Fatty cirrhosis results from the abuse of malt liquors in some cases, and is often associated with more or less obesity.

(b) **Hypertrophic Cirrhosis** (Hanot).—In most cases there is an absence of recognizable causes. Sex is a strongly predisposing cause, males being the most frequent victims, in the proportion of 6 to 1. It is not uncommon in young adults. In catarrhal jaundice the morbid processes may rarely extend to the liver and there persist, giving rise to hypertrophic cirrhosis. Cases are met with in children, in whom it may follow the acute infectious diseases. Alcohol plays an unimportant rôle in the causation of Hanot's cirrhosis. The disease is most common among the inhabitants of warm climates.

(c) **Biliary Cirrhosis.**—This form is produced by chronic obstruction of the bile-ducts (see also Obstruction of the Common Duct, p. 856).

**Symptoms.**—**Atrophic Cirrhosis.**—The symptoms of this variety of cirrhosis may present nothing characteristic as long as the sclerotic

process does not interfere with the portal circulation. In some cases the collateral (compensatory) circulation is maintained throughout the long course and symptoms fail to arise. Among the *prodromal symptoms*, a gradual loss of flesh, anorexia, constipation, a coated tongue, slight jaundice, dyspepsia, and occasionally hematemesis are to be mentioned.

As the obstruction of the portal circulation becomes more marked the mucosa of the gastro-intestinal tract becomes more and more swollen and congested, and gives rise to augmenting *nausea* and *vomiting* (most marked in the morning), and *hemorrhages* from the stomach (hematemesis) and intestines (melena), which may be frequent and profuse, but are rarely fatal. Severe hemorrhages may also occur from enlarged, varicose esophageal veins. The *tongue* is coated.

Owing to the establishment of a compensatory circulation the superficial epigastric and internal mammary veins enlarge, forming about the umbilicus the so-called "caput Medusa."

*Hemorrhoids* also are not uncommon, and are due to passive congestion of the inferior hemorrhoidal veins. As the disease progresses the *general emaciation* becomes more marked. The face assumes a pinched expression, the tip of the nose has a purple tinge from distended veins; the eyes are sunken, the cheeks hollow, and the skin presents a sallow tint (*hepatic facies*). The failure of the compensatory circulation gives rise to *ascites*, and the latter causes in many instances hydroperitoneum, leading to enormous distention of the abdomen. The *spleen* becomes enlarged. At any stage, although generally in advanced cases, *toxemic symptoms* may develop, due to some poisonous product in the blood, the exact nature of which is unknown: these are violent headache, followed by wild, noisy delirium, convulsions, stupor, and coma. They not uncommonly occur without jaundice, and have been mistaken for uremia.

*Fever* may be absent throughout the course of the disease, but is often present, and may reach 100°–102° F. (37.7°–38.8° C.).

Examination of the *urine* shows it to be of increased specific gravity, loaded with urates, and containing bile. In a small proportion of cases it is slightly albuminous, and contains casts, though out of 28 urinalyses in cases of cirrhosis Henry discovered the presence of albumin in but one. The amount of urea is constantly diminished, owing to the disturbance of the urea-forming function of the liver. An excess of indoxyl sulphate in the urine is a frequent occurrence.

The *physical examination* in a typical case of atrophic cirrhosis reveals a distention of the abdomen: there may be also an extreme enlargement of the superficial veins over the surface of the body. An icteroid tint of the skin is present in about 25 per cent. of the cases.

*Palpation* of the liver and spleen may be greatly interfered with by the large amount of peritoneal fluid present. On withdrawal of the latter, however, the spleen is found greatly enlarged and extending in some instances to the epigastric region.

The liver may show slight enlargement in the beginning of the disease; but it soon atrophies, and in emaciated subjects with lax abdominal walls its finely granular or nodular edge can be *felt* above the margin of the ribs. *Percussion* shows its vertical diameter, which normally extends from the sixth interspace to the costal margin, and averages about 4 inches (10 cm.), diminished, especially toward the median line.

Posterior dullness begins lower than normally. It must be recollected that the liver is often enlarged in otherwise typical cases.

*Fatty cirrhosis*, in which the organ is sometimes enlarged, may be latent and remain unrecognized or be discovered on the post-mortem table. In five of the six cases that have fallen under my observation, however, the symptoms closely resembled those of the ordinary form of atrophic cirrhosis, apart from the alteration in the size of the liver.

(b) **Hypertrophic Cirrhosis.**—In this variety of the disease there is usually an absence of any alcoholic history, and it is apt to be met in young adults and even children (*vide* Etiology). *Moderate enlargement* of the liver may be present before any *subjective symptoms* are observed. The latter may be absent, except the presence of slight jaundice and an occasional disturbance of digestion, until late in the course of the disease. Intense jaundice, fever, and hepatic enlargement may then appear, with the rapid development of a grave general condition. The *urine* contains bile-pigment, but the stools are not typical (pale drab or slate colored). Paroxysms of *pain* resembling hepatic colic, though less severe, may occur at irregular intervals. *Hemorrhages* into the skin from the mucous surfaces (due to passive congestion) are also common. In long-standing cases albumin and tube-casts may be present in the urine. Leucin and tyrosin have also been found, but are not constant. These symptoms are probably due to recent inflammatory infiltration arising in the course of an old cirrhosis. Splenic enlargement occurs, but ascites is exceedingly rare. The cases run an extremely chronic course, and in an instance under my care in a lad of 14 years, the grave symptoms mentioned above suddenly developed and carried off the patient after four years of slight, though decisive jaundice, and moderate hepatic enlargement. The stools were dark and bilious looking, and hemorrhages from the mucous surfaces frequently occurred; petechiæ, with urticaria and lichen, marked the skin, while pruritus was exceedingly distressing. There was a leucocytosis. Sicard and Remlinger speak of a deformity—a lowering of the shoulder, arm, and side of the chest and pelvis on the right side—but this has not been observed in any of my cases.

*Physical examination* shows a decided, and, in some cases, a uniform enlargement of the organ; the lower border is felt distinctly outlined below the costal margin, its edges being rounded and in some instances finely granular. On making deep pressure tenderness may be elicited.

*Percussion* shows an increased area of hepatic dullness, most marked anteriorly toward the median line, extending below the costal margin.

Late in the disease, in addition to the grave symptoms described above—icterus gravis, high fever, hemorrhages, and the like—serious nervous symptoms, as delirium, convulsions, stupor, and coma, may supervene. The temperature now usually ranges from 102° to 104° F.—38.8°–40° C., (*febrile jaundice*)—although fever may sometimes be absent throughout the course of the disease. Death is sometimes threatened, also, by the development of an intercurrent disease or progressive asthenia.

(c) **Biliary Cirrhosis.**—*Symptoms and Diagnosis.*—The clinical interest of this form centers principally around the symptoms of the causative condition—chronic obstruction of the bile-ducts—which have



been given in detail elsewhere (*vide* p. 856). With the latter may be associated the features, both local and general, of either catarrhal or suppurative cholangitis. *Jaundice* is usually more intense than in the hypertrophic form, particularly during the earlier stages. *Intermittent hepatic fever* is commonly observed. The *physical signs* are similar to those of Hanot's cirrhosis.

The *diagnosis* of biliary cirrhosis rests on the presence of the characteristic features of prolonged obstruction of the bile-ducts, from impaction by gall-stones, a tumor or stricture of the duct, and the like, with slow and gradual, smooth, or slightly granular, hepatic enlargement. It is to be recollected that when obstruction of the gall-ducts becomes complete, or "acute fermentative changes" are set up in the retained bile, the cases may terminate acutely (*e. g.* in acute atrophy or acute fatty degeneration).

**General Diagnosis.**—(*a*) **Of Atrophic Cirrhosis.**—An assured diagnosis may be based on the following points: 1. A clear history of the most common causes (inebriety, male sex and middle life, rickets, diabetes, gout, malaria). 2. The combined presence of ascites, with hypertrophic facies, and diminution in the size of the liver, as shown by the physical signs. 3. Absence of the characteristic features of acute disease, and the negative character of results from an examination of the heart, lungs, and kidneys. It is to be recollected that the volume of the liver is not invariably decreased in this variety, and even may be slightly increased.

With the atrophic form of cirrhosis, *chronic peritonitis with effusion* is most liable to be confounded. In the latter disease there are characteristic abdominal tenderness, fever, and usually associated tuberculous lesions of other organs (lungs, kidneys, intestines); but the hepatic facies and clearly indicative history of atrophic cirrhosis are absent. A large peritoneal effusion is in favor of cirrhosis.

(*b*) **Of Hypertrophic Cirrhosis.**—The principal diagnostic points are an absence of the usual alcoholic history, slight icterus, extending over a variable and oftentimes long period, paroxysms of pain, mucous and cutaneous hemorrhages, with moderate enlargement of the liver, and the rapid development of grave symptoms at any stage, as intense jaundice, fever, and sometimes marked nervous phenomena.

**Differential Diagnosis of Hypertrophic Cirrhosis.**—This may be confounded with *carcinoma of the liver*, *hydatid cyst*, *hepatic abscess*, and the *biliary form of cirrhosis*.

#### HYPERTROPHIC CIRRHOSIS.

Absence of recognizable causes.  
Occurs in young adults and even in childhood.  
Usually a primary affection.  
Jaundice is slight unless grave symptoms suddenly develop; there is no cachexia.  
Paroxysms of pain. The case runs a slow course, usually lasting many years.  
Enlargement is uniform.

#### CARCINOMA OF THE LIVER.

Hereditary history.  
Usually occurs after forty years of age.  
Often occurs as a secondary growth.  
Anemia is present, and also the development of a typical cachexia.  
Pain more constant with rapid emaciation. The case terminates usually within one year.  
The liver is irregularly enlarged, and contains umbilicated nodules.

(See Fig. 60.)

## HYPERTROPHIC CIRRHOSIS.

History negative as to alcohol. More common in warm climates.

Occurs idiopathically.

Fever, jaundice, and ascites may be present singly or together.

Anemia and emaciation slowly progressive. There is a leucocytosis.

Regular enlargement of the liver. No fluctuation nor thrill.

Aspiration is negative.

## MULTILOCULAR HYDATID CYST.

History of ingestion of the embryo of *tænia echinococcus* with improper food.

Simultaneous occurrence in colonies or in others in the vicinity.

No fever, pain, jaundice, or ascites.

Emaciation not marked; no leukocytosis.

On palpation an irregular, fluctuating tumor is felt over the hepatic area, giving an "hydatid thrill."

Aspiration gives a clear, serous fluid, rich in chlorids, and containing hooklets.

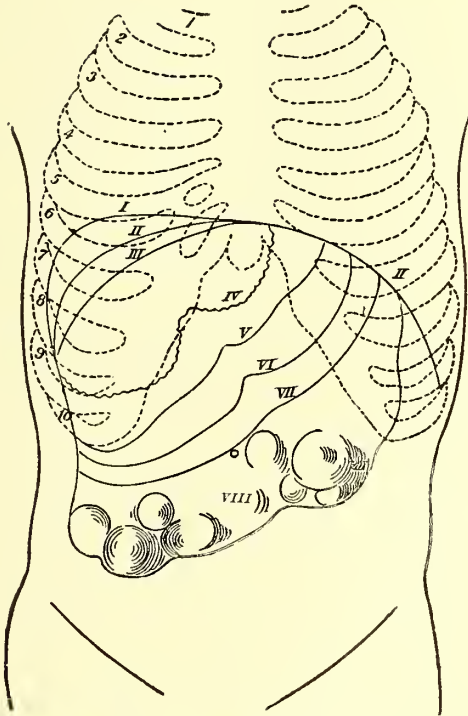


FIG. 60.—Showing approximate enlargement of the liver corresponding to the different diseases described in the text (after Rindfleisch): I, position of the diaphragm to the maximum enlargement (carcinoma); II, III, normal situation of the diaphragm; II, III, relative dulness; IV, border of the liver in cirrhosis; V, border in health; VI, lower border of the fatty liver; VII, of the amyloid liver; VIII, of carcinoma, leukemia, and adenoma.

## HYPERTROPHIC CIRRHOSIS.

Etiology usually negative. May rarely follow acute infectious diseases.

There are tenderness on deep pressure and paroxysmal pain.

Hectic symptoms absent although a continued fever may develop usually late.

Runs a slow course, lasting months or years.

## ABSCESS OF THE LIVER.

History of dysentery, traumatism, or pyemia.

Severe and constant pain; marked tenderness.

Hectic symptoms appear early (fever, chills, and sweating).

Runs an acute course, lasting a few weeks.

HYPERTROPHIC CIRRHOSIS (*continued*).

Slow enlargement, regular, or slightly  
nodulated. No fluctuation.  
Aspiration gives negative results.

ABSCESS OF THE LIVER (*continued*).

Rapid development of a fluctuating  
tumor in the hepatic area.  
The aspirating-needle reveals the pres-  
ence of pus.

So-called *fatty cirrhosis* may be distinguished from abscess, hydatid cyst, and carcinoma of the liver in a manner similar to hypertrophic cirrhosis, if one makes due allowance for its etiology, particularly the wrong use of alcohol.

(*c*) *Biliary cirrhosis* causes enlargement of the liver, but to a much more moderate extent than hypertrophic cirrhosis. In the former the symptoms of chronic obstruction of the bile-ducts are in evidence, so that jaundice is usually marked. The duration of biliary cirrhosis is, on the whole, shorter than that of hypertrophic, and the organ is more likely to undergo terminal diminution in size (atrophy).

**Prognosis.**—The prognosis of the atrophic form of cirrhosis is decidedly unfavorable, the function of the liver-cells having been impaired, although the principal source of danger is probably the ascites; and death usually takes place within a few months or a year after symptoms of portal obstruction appear. In rare cases the symptoms abate, owing to the establishment of a compensatory circulation, and may remain in abeyance for months or years.

The prospect of life is much enhanced by an early recognition and removal of the overshadowing cause—alcoholism. I have seen a few cures made in this manner. Even after the occurrence of jaundice, hematemesis, and toxic symptoms, under appropriate treatment patients have been known to enjoy comparative health for years.

**Treatment.**—The *prophylactic* treatment consists in improving the general health of the patient and in removing, if possible, the cause of the affection. Rest, graduated exercise, systematic bathing, and regular hours for eating and sleeping should be inaugurated and strictly adhered to. Alcohol, strong coffee, spices, and gastro-intestinal irritants of every nature must be interdicted. H. C. Wood states that tavern-keepers and bartenders who are unable or will not cease using alcohol may greatly prolong life by substituting hard cider for all other drinks. The *diet* should be simple and easily digestible. An exclusive milk-diet has been highly recommended (Semmla).

The *medicinal* treatment is largely symptomatic, no remedy having been discovered to prevent the formation of, or remove, the new-formed connective tissue. The chief object is to deplete the portal system and prevent, if possible, the occurrence of ascites. The bowels should be kept freely open by the use of saline purgatives (concentrated solution of Epsom salts), elaterium, or compound jalap powder. The skin is to be kept active by means of Turkish or Russian baths (under supervision), and in extreme cases by the steam bath or hot pack, employed just short of the point of exhaustion. The kidneys should also be kept active by the hydragogue diuretics, as potassium acetate, squills, calomel, digitalis in the form of the infusion, or Niemeyer's pill. Klemperer and others have also recently recommended urea as an efficient diuretic, and from 20 to 30 grains (1.29–1.94) may be given in solution. Urea acts best after paracentesis.



If the case be syphilitic in origin, potassium iodid should be exhibited in ascending doses.

Ascites, when it appears, calls for free and thorough diuresis, diaphoresis, and catharsis; and if not relieved in the course of a few days, tapping should be resorted to.

The operation of *paracentesis abdominis*, if performed under strict antiseptic precautions, is free from danger. The bladder having been emptied, a spot over the linea alba about 3 inches (7.5 cm.) above the symphysis pubis is anesthetized (preferably with a compress of cracked ice and salt), and a trocar is quickly thrust through the abdominal wall for a distance of about 1 inch (2.5 cm.). The distance is determined by the fore-finger, which is placed at the desired distance from the point of the cannula before its insertion. The patient must be in a sitting or semi-reclining position, so as to allow the ascitic fluid to collect by gravity in the lower part of the abdominal cavity. A tube having been attached to the cannula to convey the liquid to a receptacle, the trocar is withdrawn, the fluid allowed to run out, the cannula removed, and the wound closed by antiseptic gauze or a pledget of cotton. Turlington's balsam is then smeared over the site of puncture, and the abdominal binder, which has been previously applied, is tightened (*vide* Ascites, p. 927).

Complications, as cardiac hypertrophy, tuberculous peritonitis, or chronic meningitis, demand appropriate treatment; this is described in full in the discussion of the respective diseases.

## CARCINOMA OF THE LIVER.

**Definition.**—A malignant growth of the liver, occurring usually after the age of forty, and characterized by pain, progressive emaciation, cachexia, and the appearance of a nodular mass in the hepatic parenchyma. It may be primary or secondary, though the former variety is very rare as compared with the latter.

**Pathology.**—Histologically, the cells are not distinctive, being identical with those of carcinoma elsewhere; they are epithelial in character, having a small vesicular nucleus and much protoplasm. They are altered greatly by pressure, and vary in shape, being hexagonal, polyhedral, or amorphous. Large giant-cells and spots of pigment known as "brownish granules" are not uncommonly found in the cancerous mass. The so-called colloid cancers are nearly always mucoid, and the cells have undergone a mucoid change; the stroma of connective tissue surrounding the cancer-nests in some instances undergoes hyaline or myxomatous degeneration. In other instances the interstitial trabeculae completely surround the epithelial nests, which are separated by a basement membrane; to this variety the name of *adeno-carcinoma* has been given.

When examined microscopically, *medullary cancer*, either in a large mass (primary) or in secondary nodules scattered throughout the organ, is the most common variety found in the liver. On examining a liver that is the seat of carcinoma, one of two conditions usually presents itself: *First*, the organ may be apparently normal with the exception of one lobe (usually the right), which contains a dense whitish growth of firm

consistence, being distinct and sharply defined from the surrounding liver-tissue. On section the tumor is often of uniform density, bluish-white in appearance, and exudes a milk-white fluid known as "cancer-juice," which, when examined microscopically, is found to contain large, nucleated, and irregularly-shaped cells containing free granular matter. The center of the tumor may have undergone liquefaction-necrosis, with the formation of a cyst, or it may be the seat of an abscess. Various smaller nodules may be scattered throughout the organ by metastasis from the primary growth. The *second* and most common condition is secondary carcinoma of the liver, the primary lesion being situated in the mammary glands, pylorus, or the cervix uteri. The organ is greatly enlarged, as a rule. Numerous nodules are scattered throughout, and can usually be seen projecting beneath the capsule, those superficially situated having received the name of "Farre's tubercles." In the center of these nodules characteristic pits or umbilications are often present, caused sometimes by contraction of the interstitial trabeculae and sometimes by a central softening. On section they are usually grayish-white in color and of firm consistence, although cysts, hemorrhages, pus-cavities, or areas of hyaline and fatty degeneration are often found. The cells are identical with those of the primary growth, and are composed for the most part of cylindric epithelium.

In rare instances carcinoma occurs simultaneously with *cirrhosis* in the same liver, the organ presenting an uneven, nodular appearance, and being slightly increased in size and of firmer consistence than normal. When examined *in situ* the external appearance does not differ materially from that of cirrhotic liver, but on section the whole organ is found to be infiltrated with various-sized cancer-nodules surrounded by bands of cicatricial tissue. In some cases the excess of connective tissue and the amount of contraction are extreme, and the size and weight are reduced below the normal.

**Etiology.**—Among the more important predisposing factors may be mentioned—

(a) *Age.*—The disease seldom occurs before thirty-five or forty years of age, although cases have been known to occur in children. Descroizilles reports the case of a child eleven years old who died with a tumor in the right hypochondriac and iliac region, the autopsy revealing a liver studded with cancerous nodules, the nature of which was demonstrated microscopically.

(b) *Sex.*—Men are more often the victims of carcinoma of the liver than women. When occurring in the latter it is often secondary to carcinoma of the uterus or mammary gland.

(c) *Heredity* is said to be the cause of hepatic carcinoma in at least 20 per cent. of all cases, and is one of the strongest arguments in support of the diathetic theory of the disease.

(d) *Traumatism* may contribute.

(e) *Mechanical Obstruction.*—Primary carcinoma of the gall-bladder and bile-ducts not infrequently follows chronic obstruction by gall-stones.

**Symptoms.**—There may either be almost no symptoms of carcinoma involving the liver, or its manifestations may be intense and varied according to the extent and location of the growth or growths. Associ-

ated gastric symptoms, which increase as the disease advances, usually attend. A more or less marked cachexia may be the first noticeable feature. The chief symptoms may be considered in detail, as follows:

(a) *Jaundice*.—Discoloration of the skin and tissues is often by no means intense, and may be entirely absent. Harley states that true icterus was present in only 6 out of 100 cases seen by him, though few observers agree with him in his extreme view as to the rarity of this symptom. The reason given for its lack of intensity is that in the great majority of cases the growth is situated in the right lobe of the liver, and neither compresses the bile-ducts nor destroys the secretory cells of the liver.

(b) *Pain* is usually present to a marked degree, though it also may be entirely wanting. It is dull and boring in character, and localized generally in the right hypochondriac region. In some instances (as in the case of impacted biliary calculi) it may radiate to the right shoulder and the scapular region. It usually appears as the hepatic enlargement progresses, although cases of enormous-sized cancerous tumors of the liver have been known to occur without pain. The character and location of the pain are of diagnostic importance, and will be spoken of under the differential diagnosis.

(c) *Ascites*.—When the cancerous growth compresses the portal vessels, and also in cases of cirrhosis with carcinoma, obstruction to the portal circulation occurs, and results in the development of ascites. This may cause distention of the abdominal cavity to such an extent as to occlude the physical signs of hepatic enlargement. The cancerous growth may invade the peritoneum and cause an effusion. This symptom, however, is not frequent, at least two-thirds of all cases terminating without the appearance of ascites.

(d) *Fever* is usually absent until the later stages of the disease. It may then appear and rise to hyperpyrexia ( $105^{\circ}$  F.— $40.5^{\circ}$  C.), but it is usually moderate in degree, irregular, and intermittent in type.

(e) *Cachexia*.—In every case of carcinoma, at some stage of the disease, cachexia develops; when pronounced, it is almost pathognomonic.

(f) *Cerebral Symptoms*.—These may be absent throughout. In the advanced stages, however, the deleterious products in the blood, due to the perverted functions of the liver and the toxemic condition of the patient, often produce such striking symptoms as violent headache, mental hebetude, or delirium (less frequently) which may be maniacal in character. These symptoms resemble those of cholemia (*vide* Hepatic Cirrhosis, p. 887). The patient may die in sudden coma.

**Physical Signs.**—*Inspection* often reveals enlargement of the superficial veins over the abdomen, and a prominence in the upper epigastric and hepatic regions, varying with the degree of enlargement, may also be seen. In the nodular form and late in the disease, when emaciation has become extreme, elevations that are movable with respiration can be noticed beneath the skin.

On *palpation* the organ can be distinctly felt projecting below the costal margin and extending in some instances to a point below the level of the umbilicus. During deep inspiration the liver can be felt to move downward, and during expiration upward, the organ being under the influence of the diaphragmatic excursions. In emaciated subjects



the cancer-nodules are readily appreciable, and in some instances the central pits or depressions are palpable, forming a pathognomonic sign. Cancerous infiltration of the anterior margin is most easily felt, and in any enormous enlargement of the organ I have frequently detected them on the posterior surface as well. Rarely the liver is found to be uniformly large. Palpation may also show splenic enlargement, due to passive congestion.

*Percussion* shows flatness, extending in many cases in both an upward and a downward direction. In primary carcinoma (usually found in the right lobe) the area of hepatic dulness is increased irregularly downward and generally to the right. On the other hand, in secondary growths (usually massive) the nodules are oftener distributed equally throughout the liver. In such cases the area of dulness may extend across the epigastrium to the left hypochondriac region, the heart and other viscera being now displaced. Posteriorly, dulness may extend upward on a level with the fourth rib, and anteriorly downward to the iliac fossa. The organ may now weigh from 15 to 20 lbs. (6.5–9 kgms.), while the weight of cancerous livers in ordinary cases varies between 3 and 6 lbs. (1.3–2.6 kgms.).

**Diagnosis.**—In forming a positive diagnosis the family tendency, the history of primary carcinoma elsewhere in the body, the age of the patient, the localization of the pain in the right hypochondrium, the cachexia, and the progressive enlargement of the liver, with the characteristic umbilicated nodules, are the most reliable points. The appearance of jaundice or ascites, or both, is confirmatory.

**Differential Diagnosis.**—Among affections of other organs that are likely to be mistaken for carcinoma of the liver may be mentioned—(1) carcinoma of the *pylorus*; and (2) carcinoma of the *colon and omentum*. The chief diseases of the liver itself apt to be diagnosed as carcinoma are—(a) *abscess*, (b) *syphilis*, (c) *benign growths (adenomata, angiomata)*, (d) *hydatid cysts*, and (e) *hypertrophic cirrhosis*.

(1) *Carcinoma of the Pylorus.*—In carcinoma of the pylorus the physical examination frequently shows a hard nodular tumor that is most plainly outlined in the epigastric region. In a typical case, on deep inspiration, the tumor is pressed downward by the liver, but is not pulled upward by forced expiration, as in hepatic carcinoma. In many instances, however, adhesions bind the stomach firmly to the under surface of the liver, which may be the seat of secondary involvement. The absence of early nausea and vomiting and the presence of jaundice, as well as the negative results from an examination of the gastric contents, would tend to eliminate pyloric carcinoma.

(2) *Carcinoma of the Colon and Omentum.*—Secondary carcinoma of the intestine affects most frequently the sigmoid flexure. The symptoms of intestinal obstruction arise, constipation being followed by attacks of serous diarrhea due to irritation, and later by the presence of blood in the stools. In carcinoma of the liver, on the other hand, the bile-ducts may be obstructed, causing clay-colored stools, but otherwise the dejecta are normal; the seat of the nodular enlargement and pain is located in the right hypochondrium. Jaundice and ascites are absent in carcinoma of the colon. The tumor, if palpable, in the latter condition is more movable and is less under the influence of the diaphragm. It does not

give an absolutely flat percussion-note, as does hepatic carcinoma. Carcinoma of the omentum is usually secondary. The absence of small movable tumors in the umbilical, lumbar, or hypogastric regions, ranging in size from that of a pea to a walnut, aids in the elimination of carcinoma of the omentum. As the latter affection advances the abdomen becomes distended and painful to the touch, the bowels are obstinately constipated, and the physical signs reveal the presence of an effusion which, when aspirated, is generally serous, but sometimes bloody. Microscopic examination may possibly reveal the presence of cancer-cells, though their recognition is difficult. The liver, unless primarily involved, is not enlarged, and cachexia does not usually appear until late in the course of the disease.

From *hepatic abscess* the points of differentiation are—

#### CARCINOMA OF THE LIVER.

Is often hereditary. There is a history of a primary growth or chronic irritation. Occurs usually after the age of forty.

Jaundice is rare.

Fever is absent or slight.

Cachexia is present and almost pathognomonic.

Pain is dull and boring in character, and more constant.

A nodular, umbilicated tumor or tumors may be detected.

The enlargement is downward.

The duration is a few months to one year.

Microscopic examination reveals disintegrated liver-cells, cancer-nests, and in some cases the micro-organisms of supuration.

#### HEPATIC ABSCESS.

There is a history of traumatism or of intestinal ulceration, as in dysentery.

Occurs at any age.

Jaundice is sometimes present.

Hectic temperature, chills, and sweating.

Anemia may be present, but *never* cachexia.

Pain is sharp, lancinating, and paroxysmal.

A fluctuating tumor may sometimes be detected below the costal margin.

The enlargement usually upward.

The duration is usually a few weeks.

The microscope reveals pus, liver-cells, staphylococci and streptococci, the bacillus coli communis or the amœba coli.

*Benign Growths (Adenomata, Angiomata).*—Occasionally growths are detected in the liver, and may occur at any age; when these are present at or about the age of forty, they may be mistaken for carcinoma. The absence, however, of a primary growth in some one of the other viscera, together with the duration of the growth and the absence of cancerous cachexia, would tend to differentiate them from cancerous involvement. An examination of the blood may be of service, leukocytes being more common in carcinoma.

The **prognosis** is invariably fatal, the disease terminating rapidly in from a few months to a year. The most rapid course is run by secondary carcinoma of the organ.

**Treatment.**—The treatment is purely symptomatic. An easily digested, nutritious diet should be given, together with active stimulation to support the system. The pain may be relieved by the free use of morphin, given by the mouth, rectum, or hypodermically. For the nausea and vomiting that are apt to supervene the carbonated waters, cracked ice with champagne, or repeated doses of creasote (beechwood), dilute hydrocyanic acid, or wine of ipecac (2 minims—0.133—every hour until relieved) may be given. If violent delirium should occur during the later stages of the disease, cold compresses to the forehead or vertex, and bromids and chloral hydrate given in rectal enemata, may prove efficient.

## OTHER NEW GROWTHS IN THE LIVER.

(a) **Angioma, Adenoma, and Cyst.**

Occasionally, benign growths occur in the liver, and often with an absence of symptoms unless their increase in size gives rise to mechanical obstruction. One of the most common of these is angioma, which is often found in the livers of old people. Angiomata consist of tortuous and dilated capillaries in the hepatic connective tissue; they rarely attain to a size larger than a crab-apple, and usually cause no symptoms. Although most common in adults, they have been known to occur in children.

Adenomata and cystomata may also occur in the liver. They are both benign growths. The former is of the tubular variety, consisting of connective-tissue nests lined with cylindric epithelial cells. Von Bergman removed a portion of a tuberous adenoma of the liver with perfect recovery and non-recurrence of the growth.

(b) **Sarcoma.**

Of the many varieties of sarcomata, those occurring most commonly in the liver are the small and large round-celled and the melanotic variety, the latter often being secondary to sarcoma of the choroid coat of the eye. These grow rapidly, causing a widespread destruction of the liver-structure, with a change in the size and shape of the organ that is often demonstrable by palpation. E. R. Axtell reports a case in which at the *postmortem* the upper two-thirds of the liver revealed an entire absence of hepatic structure, and consisted of three tumor-masses which, on microscopic examination, were found to be small round-celled sarcomata. On section the tumor is seen to be of firmer consistence than the surrounding liver-tissue, and presents a dark, grayish-white, striated appearance. If the growth be of the pigmented variety, patches of a deep black or of different shades of pigment may be scattered throughout the mass. Metastasis is rapid and widespread (lungs, kidneys, heart, skin), as is shown by the fact that other organs are invariably found involved at the time of the growth and development of the sarcoma in the liver.

The *symptoms* are those of mechanical obstruction, and consist of gastro-intestinal disturbances due to passive congestion, edema, and ascites. Anemia and emaciation may become marked late in the disease, but cachexia does not develop. The passage of an intensely dark-colored urine (melanuria) has been noted in some cases. Secondary nodules may appear on the skin-surface.

The *diagnosis* can often be made from the primary growth (melanosarcoma of the choroid or sarcomata of the lymphatic glands) and from the rapid development of the tumor. From *carcinoma* of the liver melanosarcoma may be distinguished by the presence of ocular symptoms, particularly blindness of one eye, by the rapid widespread metastasis, the melanuria, perhaps, and by the absence of a true cancerous cachexia.

The *prognosis* is, of course, absolutely fatal, and the *treatment* merely palliative.



## X. DISEASES OF THE SPLEEN.

DISEASES of the spleen are mostly secondary to other diseases, the consideration of which embraces an appropriate description of the associated splenic disorders. The intimate relation between the spleen and blood accounts for the frequency with which this organ is involved in many of the blood-diseases.

## DISLOCATION OF THE SPLEEN.

(*Floating Spleen.*)

**Etiology.**—This may be due to the increased weight of an enlarged spleen, to tight-lacing, to relaxation of the ligaments, or to traumatism, and is met in splanchnoptosis. Carcinomatous enlargement of the left lobe of the liver caused it in a case I saw recently.

**Symptoms.**—The symptoms are vague, and are the result of pressure. By *physical examination* we discover with the touch the spleen as a mobile tumor pendant from the left hypochondrium; the tumor is superficial, blunt-edged, and notched on its anterior border, and may be replaced by the hand nearly in its normal position. On percussion over the splenic area the normal dullness is found to be absent:

In the **diagnosis** it is important to distinguish between *floating spleen* and simple enlargement, as well as between the former and *movable kidney*.

The **prognosis** is guarded as to cure, though favorable as to life.

The **treatment** must be mechanically supportive, consisting of pads and bands. Splenectomy has given excellent results.

## SPLENIC HYPEREMIA.

**Acute or active hyperemia** may be found as the result of the acute infectious diseases, giving rise to the *acute splenic tumor*, or as the result of amenorrhea, or of injuries and inflammation (*circumscribed hyperemia*). The organ is uniformly enlarged (except in the last-named cases), and is darker in color and softer in consistence; the capsule also is tense. This condition merges insensibly into *acute splenitis*.

**Chronic or passive hyperemia** is due to some mechanical obstruction of the portal circulation caused by tumors, cardiac, hepatic, and pulmonary disease, and pylephlebitis. The spleen is enlarged, firm, dark-red in color, and the capsule is somewhat thickened.

The *symptoms* are vague, and may consist of simply a sense of weight, fulness, and pressure, and some tenderness in the left hypochondrium. In cases of extravasation of blood and rupture of the spleen the symptoms of intestinal perforation, hemorrhage, and collapse may supervene.

On *physical examination* the edge of the spleen may be palpated below the margin of the ribs. The percussion-dullness is increased in area, especially downward and forward, and may encroach upon the slightly-curved umbilico-axillary "resonant line."

The detection of acute or chronic splenic hyperemia, as manifested in enlargement of the organ, is often of invaluable aid in the diagnosis of the causative disease.

The *prognosis* and *treatment* are embraced in those of the disease causing the congestion.

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### SPLENITIS.

**Definition.**—This term comprises acute and chronic (hypertrophic) proliferative splenitis and suppurative inflammation.

**Pathology.**—Next to the kidneys, the spleen is the favorite seat of metastatic inflammation and *embolic infarction*. Splenitis, due to a benign embolus originating in the left side of the heart or from the aorta above the splenic arteries, is usually circumscribed to a zone of sero-hemorrhagic infiltration about the resultant infarct. The latter is hemorrhagic at first, and later becomes particolored or *mixed*, and is of a yellow color, owing to partial fatty degeneration; still later it may become whitish and remain as a wedge-shaped (the base being peripheral), cheesy (necrotic softening), or even calcareous mass or as a fibrous cicatrix. Infection of the infarcts by pus-micrococci leads to the development of *small abscesses*, and the trabeculae surrounding the latter may give way until several abscesses or one large pus-sac may be formed.

*Perisplenitis* generally follows, and sometimes with adhesions attached to adjacent hollow organs, as the stomach and colon, through which the perforating abscess may discharge its purulent contents. An unfortunate termination is the bursting of the abscess into the peritoneal cavity; a more fortunate ending results in an external opening. In acute splenic tumor there is an active congestion, with round-cell infiltration and some proliferation of the splenic cells. The spleen is moderately enlarged, dark, soft, pulpy, and friable.

In cases of intense vascular engorgement, as in the acute splenic tumor of severe typhoid fever, intermittent fever, and epilepsy (during the paroxysm), *hemorrhagic extravasation* may occur, and there may finally be even a rupture of the capsule and a passage of the blood into the peritoneal cavity. In chronic splenic tumor there is a persistent hyperplasia of the splenic cells, and frequently also of the trabecular cells, minus the acute engorgement. *Cirrhosis of the spleen* (chronic interstitial splenitis) differs characteristically from that of other organs (as the liver and kidneys) in that there is *enlargement instead of contraction*. Added to the increase in the size of the spleen, there are in both forms of chronic splenitis thickening of the capsule, patches often of old perisplenitis, and a slaty color of the tissues, with more or less pigmentation.

**Etiology.**—The disease probably never starts primarily in the spleen itself. *Acute proliferative or hyperplastic splenitis (acute splenic tumor)* is seen as the result of the acute infectious diseases (typhoid, typhus, relapsing, malarial fevers). *Chronic proliferative splenitis (chronic splenic tumor)* occurs in connection with chronic malarial infection, splenic anemia, chronic passive congestion of the spleen, and

leukocythemia. The leukemic spleen represents a somewhat different form of chronic proliferative splenitis from the ordinary forms. *Acute suppurative splenitis* (*abscess*), either diffuse or circumscribed, is usually secondary to infectious (pyogenic) emboli, as in ulcerative endocarditis and pyemia. Again, as the result of simple valvulitis or aortic thrombosis, *embolic infarction* of the spleen may be found, which may soften and break down in abscess-formation from subsequent infection. Abscess of the spleen may also follow traumatism or the perforation of a gastric ulcer and the extension of adjacent suppurative processes.

**Symptoms.**—These are indefinite or absent in most cases. Usually there is no pain or tenderness unless perisplenitis exists. Considerable enlargement of the spleen may be attended with a *sense of weight, tension*, or *distress* in the left hypochondrium, and perhaps by slight *dyspnea*. Any *suppurative fever* present will most probably be disassociated from the idea of abscess of the spleen, provided the local signs of pus be absent. *Sudden pain* appearing in the gastric region, followed by the *vomiting* of *pus* and *blood*, in the course of an infectious disease, with *splenic enlargement*, may be due to the rupture of an abscess of the spleen. *Ascites* may also be present.

The **physical examination** may reveal some bulging on inspection, and a fluctuating tumor may be palpated. The enlargement may be sufficient to enable the examiner to feel the notch in the spleen, and also the anterior and lower borders reaching even to the umbilicus and to a level with the pelvic brim. The percussion-dulness is correspondingly increased.

**Diagnosis.**—This may be made from a consideration of the physical signs in conjunction with a study of the primary disease. In cases in which pus is suspected an exploratory puncture may clear the diagnosis. The splenic inflammation is rather an aid to diagnosis than a condition essentially needful of recognition in itself, by reason of its almost invariably secondary nature.

**Differential Diagnosis.**—Acute suppurative splenitis might be mistaken for *gastric* or *pancreatic disease*; but the previous history in the former, as contrasted with that of the latter affection, conjoined with the local symptoms that are more or less characteristic of the organ involved, will generally furnish an accurate means of differentiation.

The huge enlargements of chronic splenitis may be confounded with *hepatic, renal, omental, or ovarian growths*. Here a careful, discriminating observation of the constitutional state and of the physical signs is requisite for a diagnosis; even then it is often puzzling and difficult to attain. Care must be taken that splenic enlargement be not assumed when a *large pleural effusion* on the left side is causing the depressed lower border of the organ to be felt. Finally, *fecal accumulation in the splenic flexure* of the colon may be mistaken for moderate enlargement of the spleen. The former gives an irregular, doughy tumor, tympanites, vomiting, and a history of constipation alternating sometimes with diarrhea; there is no increase in the splenic area of dulness.

**Prognosis.**—This will depend upon the primary systemic condition in most cases. Abscess of the spleen is always a very grave complication, the main danger consisting of rupture and fatal peritonitis. Even in acute splenic tumor of a violent type there may be a hemorrhagic ex-



travasation so severe as to burst the capsule. Chronic splenitides are not in themselves grave disorders.

**Treatment.**—This is to be directed mainly at the causative condition. Quinin and arsenic are often useful in the malarial form, and the chalybeates, iodids, and ergot have been recommended for the various chronic splenic enlargements. Abscess must be treated by splenotomy and drainage. Splenectomy may be useful in certain cases of simple hypertrophy, but records show only about 20 per cent. of recoveries from the operation. The state of the patient must be well considered. Splenectomy is probably never justifiable in leukemic enlargement.

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## AMYLOID DEGENERATION OF THE SPLEEN.

(*Sago Spleen.*)

THIS occurs as a part of the cachectic condition attending amyloid or waxy degeneration of other organs (liver and kidneys). The condition develops in the course of cases of prolonged and wasting discharges (phthisis, empyema, suppurative ostitis, syphilis, chronic peritonitis, chronic entero-colitis). The spleen is, as a rule, greatly enlarged, putty-like, and rotund. The capsule is tense and glistening. There are two forms of waxy degeneration—namely, the so-called “*sago*” spleen and the *diffuse waxy* or *lardaceous spleen*. In the former the Malpighian bodies are chiefly affected and appear on section like sago-granules; in the latter the whole splenic pulp, and even the trabeculæ, are more or less degenerated, and on section the spleen appears pale, smooth, and homogeneous (boiled-ham appearance). This may be but a late stage of the “*sago*” spleen.

The *symptoms* are those of general cachexia, and the *diagnosis* rests upon the detection of an enlargement of the organ associated with evidences of amyloid disease in other organs.

The *prognosis* is unfavorable, and the *treatment* does not differ from that indicated for the underlying and causative disease.

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## MORBID GROWTHS OF THE SPLEEN.

THE principal new-growths are the granulomata, as tubercles and syphilitic gummata; also secondary carcinoma, melanotic sarcoma, and hydatid and other cysts. Lymphadenoma (*e. g.* in leukemia) may be included among tumors of the spleen (Stengel).

These affections of the spleen are all of rare occurrence, and are not readily, if at all, discoverable during life. They are of no clinical or therapeutic interest apart from the general or primary disease. It may be stated that *carcinoma* of the spleen is always secondary; it may be diagnosed by a physical examination, showing the organ to be enlarged, with the unmistakable signs of the primary carcinoma, as of the stomach. Secondary sarcoma is more common, and is recognized by an irregular enlargement and the presence of a primary tumor.

*Syphilitic gummata* of the spleen are often associated with amyloid degeneration and enlargement.

## RUPTURE OF THE SPLEEN.

THIS may occur as the result of an intense hyperemic engorgement, both in splenitis from the rupture of an abscess and from traumatism. In the acute splenic tumor of typhoid fever, in malaria, and during an epileptic paroxysm, rupture of the capsule has been known to occur on account of the extravasation of blood. The *symptoms* are usually mistaken for those of intestinal perforation with internal hemorrhage. The *treatment* is palliative.

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## XI. DISEASES OF THE PANCREAS.

## ACUTE PANCREATITIS.

INVESTIGATIONS of late years have rendered it probable that this disease is not so rare an occurrence as was formerly presumed, when it was not so readily recognized, owing partly to insufficient clinical and pathologic data, and partly to an indifference as to its existence.

## HEMORRHAGIC PANCREATITIS.

**Pathology.**—The pancreas is enlarged, usually firm, and somewhat chocolate-colored. Irregular areas show the circumscribed as well as the diffused form of hemorrhagic infiltration of the interstitial fat-tissue, with thrombosis of the pancreatic veins in some cases (Day). There is also an infiltration with round-cells of the interlobular tissues. Some cases are examples of degeneration (non-inflammatory). The adjacent tissues may also be found to be hemorrhagic, as the mesentery, mesocolon, omentum, and perinephric tissues. The gastro-intestinal mucosa may be hyperemic, ecchymotic, or in a slightly catarrhal state. Evidences of a localized peritonitis (*peripancreatitis*) are not frequent, though they should be looked for.

*Disseminated fat-necrosis* is quite commonly associated with hemorrhagic pancreatitis. Small areas of a peculiar (tallow-like) substance, ranging from the size of a miliary tubercle to that of a pea or even larger, are found scattered in the fatty interlobular pancreatic tissue in the omentum, mesentery, and sometimes in the abdominal panniculus or fat. H. U. Williams, from experimental researches, concludes that some substance in the pancreas, probably the fat-splitting ferment, is capable of causing changes similar to fat-necrosis. Flexner's experiments render it probable that the escape of pancreatic secretions into the peri- and para-pancreatic tissues is the origin of the fat-necrosis.

Mention should be made here of the fact that as the result of the infectious fevers we find the pancreas showing diffuse, parenchymatous, and granular degenerative changes. Chiari has also recently pointed out the fact that *postmortem* digestion is very frequent in the pancreas.

**Etiology.**—Most of the cases reported have occurred in men, and in persons past fifty years of age. An especial *predisposition* to the disease seems to be the result of cases of severe and obstinate dyspepsia (gastro-duodenal), alcoholism, glycosuria, gall-stones (Fitz), and trau-

matism. Hemorrhage into the pancreas may lead to subsequent pancreatic inflammation. A prolonged course of mercury has seemed to have a causal influence. It is seen occasionally *postmortem* in cases of acute tuberculosis, of the specific fevers, and of septicopyemia. The *direct cause* is probably an infection through the ducts of the gland.

**Symptoms.**—The *onset* is sudden and violent. It is characterized by *excruciating, deep-seated pain*, usually in the epigastrium or between the xiphoid and umbilicus. There are also *nausea* and *severe retching* and *vomiting*, *constipation*, and *speedy collapse*, ending fatally within a few days (second to the fourth—Fitz). The vomitus may consist at last of slimy mucus or dark blood. *Fever* is generally slight at first, though it may touch 103° or 104° F. later. *Dyspnea* and a rapid, feeble *pulse*, with *tactitation* and *marked anxiousness* or an *afebrile delirium*, may perhaps be present. In some cases there may be *diarrhea*, with thin and watery stools containing free fat. Instances may be repeated in which, owing to the coincident presence of gall-stones, there may be *jaundice* and *colicky pains* over the right hypochondrium. The jaundice, however, may sometimes be due to a considerable swelling of the head of the pancreas, which presses upon the common bile-duct. *Tympanites* occurs in a majority of the cases. *Hiccough* and *albuminuria* have also been noted. The pain in this disease, as well as the profound collapse, may be due either to a circumscribed peritonitis or to pressure upon the solar plexus.

**Diagnosis.**—This is at all times difficult, since many or all of the symptoms enumerated may be present in other affections. A careful inquiry into the previous history is important. The sudden development of an intense, deep-seated pain in the epigastrium, followed by vomiting, collapse, abdominal distention, with circumscribed resistance in the epigastrium, and the presence of constipation and slight fever, should point strongly to hemorrhagic pancreatitis. The detection of free fat in the dejections, and the discovery of scattered points of tenderness, when they occur, are also of corroborative significance.

**Differential Diagnosis.**—The temperature is apt to be higher and the pain and tenderness less localized and more constant in *peritonitis*. Fecal vomiting would indicate *obstruction of the bowel*. Here also we may determine the patency of the bowel by injection or inflation. Intestinal obstruction is of comparatively rare occurrence in the epigastrium, where the pain and distention of acute pancreatitis are localized; there are likely to be present more marked and general tympany, including the flanks, and a circumscribed distention of the intestinal coils.

In *perforating gastric or duodenal ulcer* there is a history of pain after eating, hemorrhages from the digestive tract, and of anemia or chlorosis occurring more commonly in the young female.

*Corrosive poisons* may be excluded by the history of the case and by an examination of the mouth and vomitus. *Hepatic colic* may also be excluded; the pain in this condition is intermittent, and referred more to the right side than in pancreatitis. There are also an early collapse and an absence of jaundice in the pancreatitis, as a rule.

*Acute gastro-duodenitis* is characterized by fever, by a history of injudicious eating, followed by mild inflammatory symptoms within a few



hours, and by an absence of the sudden prostration and collapse so common to hemorrhagic inflammation of the pancreas.

**Prognosis.**—Acute hemorrhagic pancreatitis in most cases ends in death. It is but fair to state, however, that in view of the ease with which the disease may be overlooked it is quite possible that certain cases of a less severe type may often recover; in these the recovery has been said to follow an entirely different affection. Osler reports a case diagnosed as one of intestinal obstruction in which abdominal section was performed and recovery followed. Thayer and Körte have also reported cases of cure in which a celiotomy decided the diagnosis.

**Treatment.**—This must needs be merely palliative and symptomatic. The treatment as for shock by the use of external heat and of warm saline solutions (by injection), hypodermics of morphin, atropin, strychnin, and of diffusible stimulants may probably be of some avail.

#### SUPPURATIVE PANCREATITIS.

**Pathology.**—The suppuration may be diffuse, with numerous small abscesses, or a single abscess may exist in the head or body of the pancreas, which may be considerably enlarged and the glandular structure extensively destroyed. The abscess may communicate with peripancreatic areas of suppuration, or it may evacuate either into various organs (stomach, duodenum, peritoneal cavity) or externally. Pylephlebitis and hepatic abscess or pyemia may follow. A disseminated fat-neerosis is not found so frequently as in hemorrhagic pancreatitis.

**Etiology.**—Most of the cases collected have occurred in adult males prior to fifty years of age. Intemperance, debauchery, gluttony, and various dietetic errors are among the *predisposing* causes. Infection takes place through the ducts, or from extension of neighboring septic foci.

**Symptoms.**—These may be *acute, subacute, or chronic*. Acute cases occur less frequently than the latter, there being a marked tendency of the disease to chronicity. **Acute suppurative pancreatitis** usually begins suddenly, with *severe epigastric pain, vomiting, hiccough, chills*, and an *irregular pyemic temperature, progressive tympanites* (at times limited to the left half of the abdomen), and perhaps acute splenic enlargement. *Constipation* may be followed later by *diarrhea*, and slight jaundice may appear. *Prostration* is generally great, and death may set in within one week from the onset.

Not seldom, however, the *course* is prolonged to three or four weeks, the symptoms persisting with progressive emaciation and final exhaustion. *Rupture* of the circumscribed peritoneal abscess, evidenced by copious dejections in which the sloughing pancreas has been found, and rapid diminution in the size of the abdomen, may take place.

Again, the onset may be less severe, and yet the case progresses steadily downward with little pain, slight suppurative fever, anorexia, anemia, and gradually increasing debility, lasting for months or even a year, and ending in anasarca and death. A pancreatic swelling is rarely *palpable*.

**Diagnosis.**—A limitation of the pain and tympany to the epigastrium, irregular fever, and the constitutional indications of suppuration are probably all that can be relied upon in arriving at a diagnosis. In fact, the diagnosis is hardly made *antemortem*.

The differentiation from *circumscribed peritonitis, perforative gastric*

or *duodenal ulcer*, and *acute obstruction of the bowel* is the same as in the case of hemorrhagic pancreatitis (*vide ante*).

The **prognosis** is fatal and the **treatment** surgical.

#### GANGRENOUS PANCREATITIS.

**Pathology.**—The pancreas may be found in various stages of necrosis, depending upon the duration of the disease. It may be a dark-brown, flabby, soft, friable, shreddy, and putrid mass, with areas of hemorrhagic infiltration and yellow softening, and surrounded by a dirty-greenish, thin, purulent, and ichorous fluid. In cases lasting for from three to seven weeks the gland may be found completely sequestered, lying in the omental cavity as a small, thin, brownish-black, shreddy, and foul-smelling detritus, soaked in a dark-colored, ichorous, and purulent fluid. The peri- and para-pancreatic tissues are usually involved with acute peritonitis. Splenic thrombo-phlebitis is commonly associated, and, as in the hemorrhagic, so in the gangrenous pancreatitis, disseminated fat-necrosis is frequently seen. The sloughed pancreas may be discharged into the intestine.

**Etiology.**—Males and females seem to be equally liable to this variety of pancreatitis, and persons past thirty years of age are most commonly affected. Hemorrhagic pancreatitis is the most frequent antecedent of the gangrenous form. The disease may result also from perforative inflammation of the gastro-intestinal or biliary tract, or from the simple extension of a catarrhal inflammation of those tracts into the pancreatic duct (Fitz).

**Symptoms.**—These are essentially the same as those of hemorrhagic pancreatitis. The *course* may last longer, however, so that death may not occur until the second or fourth week, preceded by symptoms of collapse.

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#### CHRONIC PANCREATITIS.

**Pathology.**—The pancreas is indurated from an increased development of interstitial fibrous tissue. The secreting glandular substance may be nearly obliterated, or at least considerably changed, and, owing to occluding pressure upon the duct of Wirsung, small pancreatic cysts may be formed. Interstitial hemorrhages and peripancreatic adhesions may be present. In *chronic suppurative pancreatitis* there may either be several small circumscribed abscesses or one large pyogenic cyst. The pus is often found to have undergone cheesy changes or calcareous infiltration.

**Etiology.**—Chronic pancreatitis may be due either to one or to several attacks of the acute disease. Chronic inflammation of the pancreatic duct—often secondary to gastro-duodenal catarrh—is the most frequent cause. Persistent inflammations of contiguous structures, frequent irritation from biliary calculi, and the causes of cirrhotic changes in other organs (chronic alcoholism, syphilis) probably also lead to this disease. The condition may be limited to a part of the organ.

**Symptoms and Diagnosis.**—The symptoms are hardly indicative of the disease. For a long time the symptoms of *chronic gastric catarrh*, frequently attended by *diarrhea*, may compose the clinical picture. Later there may be paroxysms of *deep epigastric pain*, and slight *fever*, with *great anxiety* and *faintness*, occurring at irregular intervals. Some *ascites* and occasional *jaundice*, due to pressure, may be observed. The detection of *free fat* in the dejections (without jaundice), and the occurrence of *glycosuria* and *lipuria*, would be of distinct diagnostic value. The presence of glycosuria in this variety of pancreatitis probably indicates an extreme degree of destruction of this gland (Fitz). A *cachectic*, emaciated appearance may be associated. *Circumscribed resistance* on palpation in the pancreatic area has been noted. Evidences of hepatic cirrhosis or of chronic renal and arterial disease may be present, and are likely to overshadow the pancreatic lesions.

The **prognosis** is grave. It is to be recollected, however, that the greater portion of the gland may become functionless, as the result of progressive fibrous change, without much impairment of the general health or the production of permanent (fatal) glycosuria.

**Treatment.**—The major treatment is *dietetic*. Fats and starches, since they demand the pancreatic ferment for their conversion, are to be interdicted, or, if permitted, are to be, so far as may be, artificially digested by the administration of tablets of pancreatin and soda (gr. v-x—0.324–0.648) fifteen or twenty minutes after meals. Malt diastase, combined with alkalies, should also be tried. Becher has found that carbonated waters stimulate pancreatic secretion in dogs, and hence their use may be advised. According to the result of Abelman's experiments, minced pancreas promotes the digestion of fats.

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## PANCREATIC HEMORRHAGE.

(*Pancreatic Apoplexy.*)

It is only in recent years that this fatal affection has been clearly isolated and defined, and mainly through the observations of Zenker, Draper, and especially Fitz.

**Pathology.**—The pancreas may or may not be enlarged; it may also be soft and friable. The hemorrhage is apt to occur into circumscribed areas of the gland, particularly its head, the interstitial and subperitoneal tissues both usually being the seat of hemorrhagic infiltration of a dark-purple color. Extensive hemorrhage may be found in the omentum, transverse mesocolon, in the retroperitoneal fat-tissue, and surrounding the kidney even. Hemorrhages into the adjacent mucous surfaces have been detected in some cases. Secondary reactive inflammations and necrosis are commonly noted.

**Etiology.**—*Slight hemorrhages* into the pancreas may be found that are secondary to excessive chronic passive congestion or to hemophilic or purpuric cases, and they may be met with in acute infective diseases. These have, however, no clinical import. The precise cause or causes of *marked hemorrhage* into the pancreas are not known. Most cases have occurred in adults past forty years of age in whom the previous health



was unusually good. Traumatism may be a direct cause. Again, some local vascular weakness or lesion (*e. g.* necrosis), superinduced by alcoholic habits or a rich diet in an atheromatous person; or, some corrosive action of the pancreatic secretion, may operate as causes.

**Symptoms.**—The patient may have been in apparently robust health when the attack comes on with *sudden* and *startling* gravity. The most prominent early symptom is *intense pain* located in the epigastric region or in the lower chest, together with a sense of *constriction*. *Nausea* and *vomiting* may be associated, and the latter is usually obstinate and gives only temporary relief. *Tympanites* may also occur. There are early and constant *general evidences of internal bleeding*—an anxious countenance, restlessness, depression, yawning, pallor, cold sweat, a lowered surface-temperature, and a small, rapid, and weak pulse. *Prostration* and *syncope* follow, and death ends the case in from half an hour to twenty-four hours. Death is caused by reflex paralysis of the heart, due either to some coincident vascular affection, or to pressure, perhaps upon the solar plexus and semilunar ganglion (Zenker).

I have collected 24 cases of pancreatic apoplexy exclusive of the 16 cases previously reported by Fitz, in which the condition led to speedy death from shock or possibly from compression of the solar plexus. Owing to its apparently “idiopathic” character, its sudden development, and quick destruction of life, pancreatic hemorrhage assumes intense medico-legal interest and importance.

**Diagnosis.**—Given the sudden-developed signs of a concealed internal hemorrhage, with pain referred distinctly to the epigastrium, and vomiting and rapid collapse, a *probable* diagnosis may be made.

**Treatment.**—This consists in relieving the pain by opiates and in overcoming the collapsed condition by free stimulation.

## CARCINOMA OF THE PANCREAS.

**Pathology.**—Primary carcinoma is the more frequent variety. It is of the scirrhus form in most cases, and usually involves the head of the gland, which may attain to the size of a child's head. Not rarely the adjacent organs are found affected, either by direct or metastatic extension of the disease. or by the pressure of the growth; the liver, peritoneum, stomach, portal vessels, bile-ducts, ureters, and aorta may thus be involved. The pancreatic duct may be occluded, so as to form retention-cysts. *Mechanically*, carcinoma of the pancreas causes jaundice and other symptoms (*vide infra*).

**Etiology.**—*Men* past forty years of age are most liable to carcinoma of the pancreas, though it has been met with even in the *new-born*. Mirallié has collected 113 cases of primary carcinoma of this viscus (Fitz).

**Symptoms.**—These are scarcely ever sufficient to indicate the disease with any certainty. There are usually a *stubborn dyspepsia*, a *progressive loss of flesh and strength*, *anemia*, and a *dull*, or sometimes *neuralgic, epigastric pain*. *Nocturnal paroxysms* of pain are common, and are often accompanied by signs of *collapse*. In some cases *vomiting* and *diarrhea* are present. The *stools* may be light in color and greasy, and may contain blood. There may also be found an abundance of *undigested*

*muscular fibers* in the stools in the absence of diarrhea; this is an incontestable proof of faulty pancreatic digestion. Among the *pressure-effects* due to carcinomatous enlargement of the head of the pancreas there may, not rarely, be *jaundice* (due to pressure upon the common duct), which persists and "is associated with an enlargement of the liver and gall-bladder." *Ascites* may appear, from pressure on the portal vein. Chylous ascites, from pressure upon the thoracic duct, has been observed in 2 cases. The inferior vena cava may be compressed, causing *dropsy* of the lower half of the body; also the duodenum, followed by *gastrectasis* or by signs of *intestinal obstruction*. Fitz points out that carcinoma of the tail of the pancreas may be a cause of *hydronephrosis* of the left kidney, from pressure upon the ureter. *Marasmus* and the *cachexia* grow from bad to worse, and emaciation may become so extreme as to permit of a satisfactory *palpation* of the *tumor*, which occupies a position near the median line above the umbilicus. Very often, however, the growth is too deep-seated to be felt, being palpable in about one-third of the cases only. *Glycosuria* may be associated.

**Diagnosis.**—Carcinoma of the pancreas is probably present in a given case in which there are rapid and progressive emaciation, deep-seated epigastric pain, muscular fibers in the stools without diarrhea, late jaundice and enlargement of the gall-bladder, and the detection of a deeply-situated, fixed, and firm tumor in the region of the gland. The quantity of indican in the urine is diminished.

*Aortic abdominal aneurysm* may be mistaken for carcinoma of the pancreas because of the transmission of the aortic pulsation to the tumor. But in aneurysm the impulse is expansile instead of to and fro, and the contact is neither so sharp nor so sudden; moreover, the cancerous cachexia is absent in aneurysm, and the history of the case may be clearly indicative.

It is sometimes difficult to differentiate a malignant tumor of the pancreas from *carcinoma of the pylorus*, of the *stomach*, or of the *transverse colon* or *omentum*; the following points will help in the differentiation of the former two:

#### CARCINOMA OF THE PANCREAS.

The tumor is deep-seated and fixed; later it becomes slightly movable. It is not associated with gastric dilatation.

Symptoms of chronic dyspepsia manifest themselves.

The vomitus is bilious; rarely contains blood; often is that of gastrectasis.

HCl is present, while there is an absence of lactic acid.

The stools contain undigested muscle-fibers. There is an absence of pancreatic secretions. The urine may contain sugar.

There is usually jaundice; sometimes ascites is present.

Inflation of the stomach shows the absence of a pyloric growth.

The course is more acute. Death may occur within a few weeks or months.

#### CARCINOMA OF THE PYLORUS.

The tumor is more freely movable, and is usually associated with dilatation of the stomach.

There are more marked gastric symptoms.

There is "coffee-ground" vomitus; it is seldom bilious.

HCl is absent from the gastric contents; lactic acid is present.

Usually the bowels are constipated, with occasional diarrhea. The stools are black after a hemorrhage. The urine does not contain sugar.

Usually there is no jaundice or ascites.

Inflation shows the presence of a pyloric tumor.

The course is more chronic, and secondary growths appear in the liver.

*Neoplastic growths of the transverse colon* are also more often superficial, and are movable and definable with the palpating fingers. There are symptoms of intestinal obstruction here, and inflation of the colon will show the relation of the tumor to the gut. In carcinoma of the colon the urine generally contains an increased amount of indican.

*Obstructive jaundice due to gall-stones* may be mistaken for pancreatic carcinoma; but in the latter affection the jaundice develops more gradually, may be less marked, and is permanent, while that of cholelithiasis is transient. In *hepatic colic* the onset is sudden and the pain is severe and colicky, or is reflected to the right and posteriorly, with equally sudden relief.

A discussion of the **prognosis** and **treatment** of carcinoma of the pancreas is obviously unnecessary.

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### PANCREATIC CYST.

**Pathology.**—Pancreatic cysts may be single or multiple, and large or small. When large they develop chiefly to the left of the median line. Sometimes a cystic pancreas may have the appearance of a bunch of grapes. Single cysts may grow to an enormous size, containing as much as several gallons of fluid. The contents may at first consist simply of retained pancreatic juice, and usually the liquid is dark gray or dark brown, alkaline, and hemorrhagic or albuminous. A hematoma may be converted into a serous cyst. The specific gravity is from 1010 to 1024. Atrophy of the pancreas may ensue. Examined *microscopically*, the contents reveal leukocytes, red blood-corpuscles, oil-drops, fatty degeneration of the epithelium, and crystals of fatty acids and cholesterolin.

**Etiology.**—Cysts of the pancreas may be due to occlusion of the pancreatic duct or its branches by compression from within or without the gland. They may also be due to tumors, to impaction of biliary or pancreatic calculi, to cirrhosis or angular displacements of the gland, or to the obstructive swelling from extension of catarrh of the bowel (Kreke). Many cases have been traced distinctly to traumatism. Lloyd suggests that the cysts that follow local injury are in reality instances of encysted peritonitis involving the lesser omentum or that portion of the latter covering the pancreas. Cysts of the pancreas usually occur in adults; Railton, however, met a case at six months of age.

**Symptoms.**—The symptoms are those of *pressure*, and in part are the result of an absence of the pancreatic secretion. *Pain* may be absent, or it may occur as colicky paroxysms, referred either to the epigastrium, the left hypochondrium, or even the left shoulder. *Jaundice* and *ascites* are present in large tumors. *Vomiting*, *constipation*, or *fatty diarrhea*, with undigested muscular fibers in the dejecta, or clay-colored, pasty, and offensive stools, may be present. *Albumin* and *sugar* may be found in the urine. *Emaciation* is not infrequent. *Intestinal hemorrhage* may occur and recur. A late and constant symptom is a *feeling of pressure* in the epigastrium.

On **physical examination** a smooth, elastic, lobulated *tumor* is discovered in the region of the pancreas if the growth is moderate in size. Sometimes a very large cyst develops in a remarkably short space of



time—*i. e.* in a few weeks. When very large in size fluctuation is easily elicited. It may be slightly movable in the grasp and during inspiration. It usually presents between the stomach and transverse colon an area of dulness, and unless the tumor be of large size it is surrounded by tympanitic resonance of deeper timber above than below. Auscultation may reveal a murmur caused by compression of the aorta. When the cyst attains enormous dimensions the usual *mechanical pressure-effects* are produced.

**Diagnosis.**—The diagnosis rests on the typical physical signs—the discovery on palpation of a smooth, elastic, lobulated, or rounded tumor that is slightly movable, and on percussion of a dull area that is not continuous above with the spleen- and liver-dulness. Resort has been had to filling the stomach with air and the colon with water (after purging), and thus proving by *palpation* the deep-seated situation (behind the stomach and omentum) of the tumor. If pancreatic fluid be obtained from the supposed cysts, it will digest albumins and emulsify fats. This test is not wholly reliable, however. A pancreatic cyst may be mistaken for an *ovarian cyst*, for *renal tumors (cysts)*, *dropsy of the gall-bladder*, and *retroperitoneal sarcoma (Lobstein's cancer)*. The differentiation is extremely difficult, and must be made by a comprehensive and careful study of all the points in the case.

The **prognosis** is good under proper treatment—incision and drainage. Of 31 reported cases thus treated, only 2 proved fatal.

## PANCREATIC CALCULI.

**Pathology.**—These are grayish-white, rounded concretions, consisting of calcium carbonate or phosphate. The calculi may be as fine as dust or as large as an almond. Among their remote pathologic effects are fistulous communications with the colon, peritoneal cavity, and stomach; also cystic dilatations of the duct and abscess-formation. Atrophy of the organ is frequently, and carcinoma rarely, associated.

**Etiology.**—Pancreatic calculi presuppose a catarrhal condition of the pancreatic duct, with retention of secretion, anomalies of the pancreatic secretion, or the presence of cysts or some other form of obstruction of the pancreatic duct.

The **symptoms** are developed when, during the passage of the stones along the duct to the duodenum, the latter excite inflammation. In consequence, paroxysms of *pain* occur (*pancreatic colic*) that are usually attributed to gall-stones, and we are often unable to differentiate the two conditions. The *radiation* of pain along the lower left costal border to the back rather than to the right side, and possibly the *detection of free fat in the stools* or *glycosuria*, may aid markedly in the diagnosis.

*Jaundice* is usually absent in pancreatic colic. Moreover, the finding of characteristic *calculi* in the stools is entirely confirmatory. *Emaciation* may become marked in calculi of the pancreas.

The **prognosis** is mainly dependent upon the associated lesions and upon certain sequelae—pancreatic cysts and chronic pancreatitis.

The indications for **treatment** do not differ materially from those of hepatic colic. Surgical intervention should be considered.

## XII. DISEASES OF THE PERITONEUM.

## ACUTE PERITONITIS.

**Definition.**—An acute inflammation of the peritoneum. The condition may be primary or secondary. Clinically, two varieties—general and circumscribed—are recognized, while, pathologically, the disease is classified according to the nature of the exudate.

*Anatomic and Physiologic Peculiarities.*—The surface area of the peritoneum is quite extensive, being almost equal to that of the skin. Fluids of all sorts are rapidly absorbed by the peritoneum, and thus, if they be poisonous, constitutional infection is speedily propagated.

**Pathology**—Upon opening the abdomen in **acute generalized peritonitis** vascular injection both of the serous covering of the intestine and of the parietal layer is observed. Even in the most recent cases the coils of intestine may be feebly glued together by lymph, while in those of longer duration the adhesions are quite firm. As in the analogous inflammation of the pleuræ or pericardium, we distinguish the following forms pathologically: (a) A *plastic* or *fibrinous*, in which there may be also a small amount of serum present. (b) *Sero-fibrinous* (inflammatory ascites), chiefly characterized by considerable sero-fibrinous fluid; additionally, the coagulated fibrin forms a covering for the parietal and visceral layers of the peritoneum. (c) *Purulent* (most frequent). The amount of inflammatory exudate varies greatly, and is frequently enormous, exceeding 30 liters (quarts). Putrefactive decomposition of the pus may occur, especially in cases due to gangrene of the gut or to puerperal peritonitis (violent forms), giving rise to a thin fluid that is grayish-green in color, is sometimes distinctly sanious, and ill-smelling. Offensive gases are present with relative frequency. These may come from the intestinal canal, following the track of perforations; or they may be due to decomposition of the purulent exudate. (d) *Hemorrhagic*. This form is common in cases that are of a cancerous or tuberculous nature, and in subjects whose vitality has been lowered by various other affections. It may also be of traumatic origin.

*Changes in the Intestines.*—The effect of acute peritonitis is to thicken the coats by inflammatory edema; soon the musculature is paralyzed. An associated catarrh of the mucosa of the intestine is sometimes observed.

The different pathologic varieties above described may be limited to definite portions of the peritoneal sac, when they are termed “encapsulated” or **localized acute peritonitis** (*vide supra*). In localized purulent peritonitis further extension of the process is arrested by the rapid formation of circumscribed adhesions due to the exudation of lymph; there are also undoubted instances of circumscribed, aplastic peritoneal abscesses. The milder forms of limited plastic and sero-fibrinous peritonitis pursue a slower course than the purulent variety, and commonly lead to the development of firm adhesions (*adhesive peritonitis*). Since the histologic changes in acute peritonitis do not differ from those observed in other inflammations of serous membranes, the reader is referred to the section on Pleurisy (p. 541) for their consideration.

**Etiology.**—The irritants causing acute peritonitis may be—(a) Or-

ganized inflammatory agents (organic irritants). These may be *specific* or *non-specific*. Among the non-specific agents are the pyogenic bacteria. Grawitz has shown that the latter can only cause peritonitis under certain conditions: they excite the disease when injected into the peritoneal cavity or when poured out from the diseased or injured membrane more rapidly than the peritoneal tissue can dispose of them; also when the epithelial layer has from any cause been removed. Absorption may be interfered with, while the pyogenic micrococci continue to enter from the bowel or other viscera in great numbers. Unfortunately, the clinical practitioner often meets with cases of peritonitis in which these pyogenic organisms are the only positive agents. These essential conditions obtain when the membrane is wounded by the perforation of gastric and intestinal ulcers, and also in perforation of the gall-bladder, in rupture of the liver, kidneys, and spleen, when the latter are the seat of abscesses, and, with uncommon frequency, in appendicitides, in purulent inflammation of the ovaries and of the Fallopian tubes. "There are instances in which peritonitis has followed rupture of an apparently normal Graafian follicle" (Osler). These perforative forms of peritonitis are at the same time the most serious and the most important. "Death may result from the injection into the peritoneal sac of putrid liquid if the dose be large enough; but it is practically the same whether the fluid is injected into the blood-stream at once or allowed to find its way into the peritoneal cavity, and the result follows nearly as quickly in the one case as in the other" (Moullin). The rapid absorption of liquid substances gives full opportunity for the phagocytic action of the white blood-corpuscles.

Among specific organic irritants the *tubercle bacillus* deserves especial mention, though, as before intimated, a discussion of its characteristics is not in place here. The *streptococcus pyogenes* is probably responsible for the most violent forms of peritonitis (*e. g.* those occurring in puerperal sepsis and post-operative varieties). The *staphylococcus pyogenes aureus* (or *albus*) has also been found in such instances.

The *bacterium coli commune* (always present in the intestinal tract) is frequently the leading factor in peritonitis of intestinal origin, and, I believe, also in that form following operations upon the appendix. Occasionally other organisms, as the *pneumococcus*, the *bacillus of Friedländer*, or the *bacillus pyocyaneus*, *typhosus* and *proteus*, the *gonococcus*, and the *anthrax bacillus*, have been found.

Multi-infection is quite common. Some contend that all forms of peritonitis are due to bacteria or their toxins.

(b) **Chemical Irritants.**—These are rather numerous and varied, though all produce their effects in one of two ways. First and most frequently, the irritant acts upon the membrane, exciting an exudation of lymph. In this instance constitutional intoxication is secondary. Secondly, the chemical irritant may be quickly absorbed and produce systemic intoxication immediately.

(c) **Mechanical irritants**, as, for example, a hernia, which may produce a localized peritonitis.

(d) Peritonitis may be due to a direct extension of infective processes from the intestinal tract or other adjacent organs. Doubtless the bacteria often penetrate the intestinal wall and gain the peritoneum by way of the lymph-channels. In the majority of instances this variety is pro-



*tective* in character and results in local adhesions. I have seen a few undoubted instances of peritonitis secondary to pleurisy in which the irritants penetrated the diaphragm along the course of the lymphatics.

(e) The disease very rarely occurs *idiopathically*: It has been attributed to exposure to cold or wet (rheumatic peritonitis). These so-called idiopathic cases are probably instances of cryptogenetic infection. As in other inflammations of serous membranes, so peritonitis may be secondary to chronic Bright's disease. In such cases the special irritants reach the membrane either from the intestinal canal or through the general circulation.

**Clinical History.**—The symptoms are both of a *local* and a *general* nature. In *sthenic* cases of perforative peritonitis they occur simultaneously with great severity and suddenness. On the other hand, in *asthenic* cases, such as occur frequently in those already afflicted with some serious disease that is apt to result in perforation (for example, typhoid fever), both the local and constitutional symptoms are more or less overshadowed by the disturbances due to the primary affection. Again, circumscribed abscesses of the peritoneum often lead to diffuse suppurative peritonitis, and the change may take place so insidiously as to defy detection. These anomalies from the typical onset and course of the disease are by no means exceptional, and should ever be distinctly borne in mind by the physician.

**Local Symptoms.**—Among these, *pain* is the chief. At the commencement its seat of greatest intensity corresponds, in most instances, with the seat of origin. Hence the character of the causal disease is often betrayed by the location of the chief pain. For instance, if this appears in the region of the stomach and is referred to the back or shoulders, we would think of gastric ulcer; if in the ileo-cecal region, of appendicular disease; and so on. It follows that quite commonly the severest pain is in the lower half of the abdomen. It is almost constant, increases in severity, and finally becomes general and excruciating; it is also much increased by deep respirations, by pressure, and by bodily movements. It remits, but does not intermit, though it may be slight in *asthenic* cases. Here the patient is excessively weak, while his sensibilities are greatly blunted. Gastro-intestinal symptoms are prominent, more particularly *vomiting*, which occurs early and is apt to recur with comparative frequency. It may follow the taking of food, though, in my own experience, it has more frequently taken place spontaneously; the *vomit* then consists of a watery liquid greenish in color and contains mucus. In rare instances the matter vomited is a dark-brown liquid. Vomiting may sometimes be absent, however, owing to the presence of marked *asthenia* or *coma*. *Eructations* are common, and *constipation* is usually present and may become exceedingly obstinate. On the other hand, there may either be *diarrhea* throughout the disease, or this symptom may precede the constipation. Constipation is due chiefly to paralysis of the musculature of the intestine. It is to be ascribed to an increased peristalsis due to intestinal catarrh. The *apex* of the heart is elevated; the *tongue* at first is furred and moist, and later it is dry, brown, and often fissured.

**Constitutional Symptoms.**—At the onset the patient in *sthenic* cases is seized with a *rigor* that may be repeated. The *shock* sustained by

the nervous system in acute peritonitis is most intense; the *temperature* rises immediately, though it does not, as a rule, attain to a high level, and it frequently presents a curve more or less characteristic of suppuration. The rectal temperature is often relatively high; the respirations are shallow and much accelerated, ranging from thirty to forty per minute. We have, as factors to account for this increased frequency, (*a*) a crowding upward of the diaphragm, (*b*) the greatly enfeebled heart, and (*c*) the pain occasioned by throwing the diaphragm into action. The *heart* early becomes excessively weak, and, as would be expected, the *pulse* is rapid, small, and soft. The pulse toward the close becomes exceedingly frequent (130 to 150 beats per minute) and is almost imperceptible; during the early stages the pulse ranges from 100 to 130. Other evidences of more or less marked *circulatory collapse* soon manifest themselves. The patient wears an anxious facial expression, the eyes are sunken, the features pinched and cool, the lips cyanotic, and the extremities are likewise cold and somewhat livid. The patient invariably assumes the *supine position*, with the lower extremities drawn up, so as to lessen the tension of the abdominal muscles, and thus to secure the greatest possible comfort. The *urine* is scanty and high-colored, and contains indican. There may be a retention of urine; though oftener, perhaps, micturition is more frequent than in health. Marked *nervous symptoms* do not appear; indeed, the mind usually remains quite clear to the close. Moderate delirium, however, which sometimes gives way to mild stupor, is met with occasionally. In connection with these facts it should be pointed out that in the *asthenic form* of acute peritonitis the constitutional features differ from those above described. The *temperature* is usually subnormal (except in the rectum), the pulse is exceedingly feeble and running, and the signs of collapse are well marked from the onset.

**Physical Signs.**—*Inspection* reveals the gradually increasing abdominal distention, that frequently becomes excessive if the intestinal walls are more or less completely paralyzed. Often the amount of effusion soon becomes large, when the abdomen appears widened. The degree of distention bears a definite relation to the severity of the inflammatory process, and is in inverse ratio to the development of the abdominal muscles. Thus, when the latter are poorly developed or greatly relaxed the expansion is enormous. On the other hand, when they are strong the muscles are apt to be quite tense, permitting of a relatively slight enlargement; the abdomen may even show a small concavity, in which case the walls are of a board-like hardness. The cardiac apex-beat is displaced upward and outward, occupying the fourth interspace.

*Palpation* elicits extreme tenderness, more particularly in the vicinity of the umbilicus. In not a few instances of acute peritonitis have I been able to detect a distinct friction-rub. *Percussion* gives at first an exaggerated tympanitic note. There is often an absence of liver-dulness in the mammary line, and rarely also it is absent in the mid-axillary line. In pneumo-peritoneum, resulting from perforation of the gut or stomach, we often meet with an absence of liver-dulness, especially when a large purulent effusion coexists. Again, a great diminution in, or even the total effacement of, the dull area may be caused by coils of intestine forcing their way up between the anterior surface of the organ

and the inner surface of the abdominal wall. Owing to the fact that the diaphragm is pushed up, both the upper and lower lines of hepatic dulness are correspondingly higher than normal. When air is present within the abdominal cavity and the patient lies upon his right side, splenic dulness disappears from displacement by the air. The lower level of cardiac dulness is as high as the fifth rib.

By means of *percussion*, sooner or later, fluid effusions are usually detectable in sthenic cases. On the other hand, there may be in markedly asthenic cases an amount of liquid exudation present that is often too small to admit of detection. When the effusion is considerable in quantity there is dulness on percussion over the most dependent parts; when tympanitic distention is excessive, however, even a copious effusion may be so effectually hidden as to elude discovery in this way. I have elsewhere reported one such instance.<sup>1</sup> On account of the painful character of the illness the patient's position cannot, in the majority of instances, be changed. When, however, the decubitus can be altered the line of dulness will be found to be movable, but the degree of mobility varies exceedingly, depending upon the extent of the peritoneal adhesions present. The effused material is partly contained in pouches, giving rise to areas of circumscribed dulness, and these must not be mistaken for the lesions of a localized peritonitis.

**Course and Prognosis.**—Asthenic forms of diffused peritonitis are perhaps invariably fatal. Though the local symptoms and signs are not marked, the characteristic evidences of collapse or of general septicemia appear and grow in intensity to the end. The *duration* in sthenic cases rarely exceeds one or two days; in asthenic cases it is longer, lasting from four or five to six or eight days. Death sometimes occurs quite suddenly, owing to cardiac exhaustion or primary shock. Although most instances are dynamic in the early stages, acute diffuse peritonitis assumes a markedly adynamic form in the later stages. The clinical peculiarities and the course of an individual case are greatly influenced by the etiology. Acute generalized peritonitis arising from perforative appendicitis, from perforation of a gastric ulcer, puerperal sepsis, or from external injuries, is usually of a violent form and ends fatally. Prompt operative intervention, however, is powerful in saving life in a small percentage of the latter class. When the disease is traceable to rheumatism or exposure recovery may take place. A case occurred in my own practice in which acute sero-fibrinous peritonitis with considerable effusion was associated with acute articular rheumatism and organic lesions of the aortic segments; the patient recovered.

Acute generalized peritonitis may not infrequently merge into a chronic condition; this, however, will receive separate consideration in its proper place.

#### LOCALIZED OR PARTIAL PERITONITIS.

(*Circumscribed Peritonitis; Visceral Peritonitis.*)

This is a localized form of inflammation of the peritoneum that is coextensive only with the serous covering of single organs, and involves a limited portion of the membrane. Hence, to the various forms of cir-

<sup>1</sup> *International Medical Clinics*, vol. iii., second series, p. 82.



cumscribed peritonitis such terms as perihepatitis, perisplenitis, perinephritis are applied. The condition is found in its most important form in *appendicitis*, but the points that are characteristic of localization in this disease have been mentioned elsewhere (*vide* *Appendicitis*, p. 819). Localized peritonitis may also be caused by a carcinomatous growth.

*Pyo-pneumothorax subphrenicus* is the term applied to a circumscribed peritoneal abscess containing air, situated between the liver and diaphragm. The condition is described under the heading *Acute Perihepatitis* (p. 875).

*Local pelvic peritonitis* (perimetritis) is the most frequent variety, and is secondary, as a rule, to inflammation about the uterus, Fallopian tubes, and ovaries. Its consideration, however, must be left to special works on gynecology.

**Symptoms.**—The *local* clinical features do not differ from those described under the diffuse form, but their area of distribution is more or less strictly limited to definite regions. By eliciting the *physical signs* with care fluid collections are sometimes demonstrable. The *constitutional symptoms* are likewise similar in character, though less marked than those belonging to the diffuse variety. There may be *rigors*, and *pyemic symptoms* appear, together with the temperature-curve peculiar to this condition. The danger of involvement of the general peritoneal cavity as the result either of rupture or of an extension of septic inflammation is a constant menace. When the peritonitis remains localized these cases may pursue a subacute or even a chronic course, though in most instances the constitutional disturbance becomes grave at last.

**Diagnosis.**—In attempting to diagnosticate acute generalized peritonitis it is of great importance for the clinician to keep in remembrance the *sthenic* and *asthenic* forms of the affection. The character and gravity of the symptoms, both general and local, are such as to render the diagnosis of the *sthenic* form entirely easy. Especially valuable features are the *constant pain*, the *marked tympany*, the *excessive tenderness under pressure*, and the *vomiting at intervals of a greenish fluid material*. Of equal importance are the serious general disturbance previously depicted, and in particular the *cool, sharpened features* and the *ever-increasing weakness and rapidity of the pulse*. These clinical manifestations clearly foreshadow cardiac exhaustion or fatal collapse. When the cases are not seen until the advanced stage has arrived, however, the diagnosis presents many difficulties. Nothing is now more important than the consideration of the history from the time of onset, also of the previous history, with a view to determining the point of origin and the probable cause of the disease (usually some such primary disease as *appendicitis* or *gastric ulcer*).

The smaller number of cases belonging to the *adynamic* type are from the start extremely difficult of diagnosis. Here a history that is clearly indicative, the presence of moderate tenderness, and augmented tension of the abdomen, with profound collapse, would point to this condition. It must, however, be confessed that a positive opinion is often unwarranted, owing to the absence of the more characteristic clinical indications.

**General Differential Diagnosis.**—*Hysteric peritonitis* (so-called) simulates in every leading particular the genuine form so closely as to make the distinction an insurmountable difficulty, unless there be present other hysteric manifestations. In my experience the tenderness has been out of proportion to the gravity of the constitutional disturbance. The patient often complains bitterly before the abdomen has been touched; on the other hand, when his attention has been otherwise engaged firm and prolonged pressure can be made.

Acute generalized peritonitis occasionally supervenes on *typhoid fever*. In such cases it is caused either by perforation of the intestine or by a direct extension of inflammation from a deep typhoid ulcer. If consciousness be retained, sudden severe pain, tenderness followed by excessive tympany, and signs of collapse will establish the diagnosis. Peritonitis, however, develops more often in those grave cases of typhoid that are attended with coma, marked meteorism, and profound adynamia, and under such conditions it often remains unrecognized (*vide Typhoid Fever*, p. 35).

In *acute enteric catarrh* the meteorism and sensitiveness under pressure are usually less pronounced; the disease also lacks the marked constitutional symptoms of acute peritonitis. The pain is colicky, is characterized by exacerbations, and even intermits in entero-colitis, while it is constant in peritonitis. The pain in acute enteric catarrh is often followed by diarrheal stools.

*Intestinal colic* is distinguished from peritonitis by the flatulence, the borborygmi, and the wandering pain in the absence of all other phenomena.

*Rheumatism of the abdominal muscles* excites pain, which, however, is superficially located (the disease being an affection of the muscular layer), and is frequently associated with rheumatism in other parts of the body. There may also be a clear history of previous rheumatic attacks.

*Tubal pregnancy (after rupture)* has also been confounded with acute peritonitis, but its differential diagnosis is fully discussed and must be looked for in special works on gynecology and obstetrics.

*Rupture of an abdominal aneurysm* and *embolism of the superior mesenteric artery* are also conditions that give rise to peritonitic symptoms—meteorism, recurrent vomiting, and collapse—all appearing with explosive violence.

Acute generalized peritonitis in its symptomatology bears a close resemblance to acute intestinal obstruction, and the discriminating points have already been tabulated (*vide* p. 831).

**Prognosis.**—This is less grave than in the diffused form, and recovery may often be expected. Timely surgical intervention, particularly if a tendency to spreading be shown, may render the outlook encouraging or even lead to prompt recovery.

*Sequelæ.*—If recovery should take place, the inevitable result is the formation of adhesions and fibrous bands, the contraction of which may cause constriction of the bowels, bile-ducts, and other structures.

**Treatment.**—*Hygienic and Dietetic.*—The patient should be placed in the position that will give him most comfort, and should be kept absolutely undisturbed. The sick-room should be of good size and well ventilated; the temperature should be kept at from 65° to 70° F.

(18.3°–21.1° C.). The diet demands careful attention. Pancreatized milk in accurate dosage (5iv–vj—128.0–192.0—every two hours) should be administered, and if the stomach will not bear the introduction of nourishment, recourse should be had to rectal alimentation. Other liquid food-stuffs, as meat-juices and egg-white (diluted), may also be allowed. In asthenic cases alimentation must be generous, although solid articles of food are to be avoided.

**Medicinal.**—Formerly the opium method of treatment, first instituted by the late Alonzo Clarke, was the one followed by the bulk of the profession. His plan was to administer  $\frac{1}{2}$  gr. (0.0324) of morphin or its equivalent (gr. ij—0.129) of opium, and repeat the dose every two hours until the respirations were lowered to ten or twelve per minute. The pupils were then observed to be contracted, the pulse from 76 to 80, the pain relieved, and peristalsis arrested. This latter effect was obtained, even though in the case of some patients larger doses of opium than here indicated were necessary; in others smaller doses sufficed. The bowels were absolutely let alone. It is explained that in favorable cases the bowels moved spontaneously at the end of one week, and that the patient then entered upon convalescence. This method of treatment is at present adhered to only by the ultra-conservative element of the profession. Among those authors who recommend opium as the most efficient measure in the treatment of this disease many still advise against the immoderate dosage previously so generally administered, but employ just enough to keep the patient well under the influence of the drug.

The leading mode of treatment to-day consists in the use of *saline purgatives*, exhibited in divided doses in concentrated solution (3j–ij—4.0–8.0—every two or three hours) until the irritating intestinal contents, should any be present, are removed, and additionally several copious serous discharges occur daily. Purgatives do good when given in this manner principally by causing a rapid exosmosis of serum from the blood-vessels of the intestines, by removing the collateral edema, and by indirectly relieving the congestion of the peritoneum, thus promoting a rapid absorption through the latter membrane. By increasing the peristaltic movement they also diminish the danger of peritoneal adhesions. The remedies to be selected will depend upon two primary considerations: *first*, the etiology of the individual case (whether a communication has or has not been established between the peritoneal cavity and the bowel), or an intra-peritoneal abscess or abscess-cavity in one of the abdominal viscera; and *secondly*, the type of the case, whether sthenic or asthenic. If perforation is known to have taken place or the occurrence of this accident is strongly suspected, a prompt laparotomy, followed by the free use of salines, is the proper treatment. After the *primæ viæ* have been looked after by the surgeon, salines, for the reasons before stated, are to be used with a free hand. For a like reason they are most serviceable in peritonitis due to extension of the inflammation, and also in the puerperal form. If the patient be robust, with a full, tense pulse, we may begin the treatment by the use of mercury, the best preparation being calomel, exhibited in fractional doses (gr. ss—0.0324—every hour) until its purgative action is obtained; this is to be followed by the salines. The object of the *calomel treatment* is to de-



fibrinate the exudations as well as the blood of the patient. Certain observers advocate the use of small doses of calomel, and seek to avoid any purgative action of the remedy. Indications demanding the *opium treatment* do not often present themselves. When, however, the vital forces are profoundly depressed, as shown by the symptoms of collapse, and there is not even a reasonable suspicion of perforation, then opium should be tried, but not in the heroic doses formerly advocated. Enough only should be given to obtain the physiologic effect of the drug in a moderate degree. Again, if the evidences of perforation into the general peritoneal cavity are complete and competent surgical skill is not at hand, large doses of morphin are imperative, with a view to relieving pain, keeping the patient at absolute rest, and sustaining the heart against the exhausting effect of shock. The bowels should now be relieved by simple large enemata. The value of serum-therapy in this disease is as yet uncertain (Fowler).

**Local Treatment.**—At the onset, if the patient be strong, from twenty to thirty leeches are to be applied to the abdomen. The ice-bag or ice-poultices are often of distinct service in the earlier stages. Later, in localized peritonitis, blisters may be useful, although objectionable in the event of surgical intervention becoming necessary. In cases in which meteoric distention is not great I have also made repeated trial of an ointment containing ung. ichthyol (ȝij—32.0); ung. belladonnæ (ȝss—16.0); ung. hydrarg. (ȝij—64.0); this is applied to the entire abdomen thrice daily.

In order to relieve the *tympany* turpentine stupes are serviceable. I have also had favorable results from the insertion of the long rectal tube (soft esophageal) well up in the colon. *Large* high enemata should be used; and turpentine combined as follows may prove efficacious:

R̄. Turpentine,	ȝij (8.0);
Ox-gall,	ȝij (8.0);
Milk of asafetida,	ȝiv (128.0);
Warm water,	ȝvj (192.0).

Puncturing the abdomen with a hypodermic needle in order to relieve tympany, as recommended by Loomis, may also be resorted to, though I have had no personal experience with this measure.

*Pain.*—No matter what general plan of treatment is pursued, the pain must be relieved by opium in some form. *Thirst* is to be relieved by chipped ice, over which a little brandy may be sprinkled. The *vomiting* is best treated by carbonated water exhibited in small quantities, or by iced champagne similarly administered. One-drop doses of creosote are also of value. For the systemic collapse, as well as for combating thirst and vomiting, I can warmly recommend saline infusion, to be repeated if needful.

## CHRONIC PERITONITIS.

**Definition.**—Chronic inflammation of the peritoneum.

**Pathology and Etiology.**—The anatomic characters presented by different cases are greatly varied, though for convenience of study they may be considered under two divisions (as in the acute form): 1. *Local*; 2. *General*. The latter may be (a) **Adhesive**, when the peritoneal layers are inseparable and indistinguishable, with an obvious thickening, and the intestinal coils are everywhere seen to be grown together. The cause is usually a previous acute attack, and, doubtless with great relative frequency, the condition is produced by the *acute progressive form* (Mikulicz), which is *localized* at the start. Rheumatism is also an occasional factor, and a mild variety of adhesive peritonitis, confined, as a rule, to small circumscribed areas, may be engendered by the trocar used for tapping in ascites.

(b) **Proliferative Peritonitis.**—“The essential anatomic feature is great thickening of the peritoneal layers, usually without much adhesion” (Osler). It has been found to be associated with cirrhosis of the stomach, liver, and other abdominal organs. The amount of liquid effusion, varying in composition from serum to pus, is usually moderate, and it may, owing to adhesions, be loculated. The omentum is sometimes rolled up in the form of a massive cord, with its long axis in the transverse direction. In an autopsied case of chronic peritonitis apparently secondary to hepatic cirrhosis I observed in the thickened membrane numerous small hard nodules that were at the time regarded as being tuberculous in nature. It is to be pointed out, however, that a number of cases of pseudo-tuberculosis have been recently reported. In several of these an operative incision was followed by recovery, and this was put down as a cure of tuberculous peritonitis till the microscope showed the nodules to be fibrous. Among *etiologic factors* chronic alcoholism stands first. In one case that I saw, acute followed by chronic rheumatism seemed to be the only assignable cause. The condition is sometimes secondary to chronic nephritis, to syphilis, or a general fibroid process.

(c) **Cancerous Peritonitis.**—Quite often in connection with cancerous growths in the peritoneum a well-marked peritonitis is evident. There may be a liquid exudation, which is apt to be bloody and chylous.

(d) **Chronic Tuberculous Peritonitis.**—This is the most important variety. The inflammatory lesions are quite pronounced, as a rule, and lead to marked thickening of the layers—changes that are to the naked eye identical in appearance with those noted under the preceding forms, but which on histologic examination show the presence of tubercles and caseous degeneration. The amount of liquid effusion varies within wide limits, and is usually blood-stained. The frequent association of hepatic cirrhosis with tuberculous peritonitis should be remarked. From tuberculous peritonitis, tuberculosis of the peritoneum is also to be distinguished clinically; the latter may be acute or chronic, and the lesions consist in the deposit of various sized tubercles without much collateral inflammation. Acute and chronic tuberculosis of the peritoneum have received due consideration in their appropriate place (p. 309).

(e) **“Chronic Hemorrhagic Peritonitis.”**—This term should be limited in its application to that form first described by Virchow, in which the

peritoneum is at intervals partly covered by a membrane of new connective tissue that alternates, as it were, with layers of hemorrhagic extravasation. A similar condition results from the frequent use of the trocar for ascites.

**Chronic Localized Peritonitis.**—This is of frequent occurrence, and is confined most commonly to the serous covering of the spleen, liver, and certain portions of the bowel, particularly of the appendix. The condition results in the formation of *firm adhesions*, with matting of the intestinal coils and fibrous bands. It is usually the *sequel* of localized acute peritonitis occurring in connection with inflammatory diseases of the different abdominal organs.

**Symptoms of the General Forms.**—Whether chronic peritonitis follows the acute form or not, it always develops insidiously. Most cases remain quite obscure, and not a few are totally devoid of clinical manifestations. The patient may complain of disorders of the *alimentary tract*, and especially of *constipation*. On the other hand, *diarrhea* is observed in tuberculous peritonitis from associated intestinal ulceration. Rarely pressure, from the traction force of the adhesions, on the common duct or portal vein gives rise to obstructive *jaundice*, or *ascites*, as the case may be. I saw an instance recently in which compression of the veins leading to the lower extremities caused unilateral *edema*. *Subjective abdominal sensations*, as uneasiness, oppression, heat, and pain (often colicky in character), are experienced. Sometimes pain is entirely absent.

*General symptoms* appear, though they are quite vague as a rule. An irregular fever, hectic in type, is occasionally observed. Later, increasing general weakness, emaciation, and general nervous disturbance become rather prominent clinical features. Some of these phenomena, however, may be due to associated affections. When the peritonitis is tuberculous we frequently see clinical evidence of the causal disease in other parts of the economy (*vide* Tuberculous Peritonitis, p. 309).

**Physical Signs.**—*Inspection* usually shows the belly to be slightly, though unequally, enlarged. As in acute peritonitis, so here, we find the belly flat, or even concave occasionally, with great tension of its walls. Fluctuation is sometimes obtainable over limited areas only, since the fluid is not free, but encapsulated. The coiled-up and shrunken omentum may be *palpable* as a sausage-shaped mass, and thick bands of adhesion may also not rarely be felt, in different places, as hard, uneven masses simulating neoplasms. The *percussion-dulness* varies considerably with the amount of effusion, its arrangement, the degree of peritoneal thickening, as well as with the character and locality of the fibrous bands. It follows that in some cases irregular areas of tympanitic percussion-resonance and of dulness are to be found side by side scattered over the abdomen. Obviously, too, changing the patient's posture would not give movable dulness, owing to sacculation of the fluid. A marked sense of resistance is experienced on percussion over the dull area. *Friction-fremitus* can sometimes be elicited, and less frequently *friction-sounds* also during forced breathing.

**Symptoms of Chronic Local Peritonitis.**—This condition is often entirely latent. When not so, the most characteristic indication is constant *pain*, distinctly colicky in nature and often quite intense.



The physical signs are negative, as a rule. Very rarely a resistant, ill-defined mass, corresponding with the seat of greatest pain, can be felt. A fibrous band may be so arranged as to form a snare through which a knuckle of bowel may pass, with resulting strangulation. Fitz's analysis of 295 cases showed 63 to be caused in this way.

**Diagnosis.**—That form of chronic peritonitis (serous or granular) most frequently seen in females at the commencement of puberty is hard to discriminate from *tuberculous peritonitis*, since the latter may be more or less latent. Tuberculous peritonitis is attended with fever, more pain and tenderness, and there is a more rapid accumulation of the exudate. Again, the general features, debility and loss of flesh, progress more rapidly than in granular peritonitis. The detection of conclusive evidence of the disease in persons closely related, or on physical examination of associated pulmonary or pleural lesions, would render the diagnosis of tuberculous peritonitis almost certain. In obscure cases the guinea-pig should be inoculated with the exudate (see *Pleurisy*, p. 541).

**Course and Prognosis.**—The milder varieties of simple chronic peritonitis may, though rarely, reach a favorable issue. In cases belonging to this category the disease takes a chronic course, and leads gradually to a condition of extreme debility, even if it does not, as is usually the case, materially shorten life. Tuberculous peritonitis has, until recently, been regarded as being almost uniformly fatal at the end of several months. Cures that must be attributed to the surgeon's work, however, are at present by no means uncommon. Rarely spontaneous cures also occur, particularly among children, in whom the disease is less serious than in adults.

**Treatment.**—The patient should be enabled to enjoy the benefits of good sanitary surroundings. Close attention is to be paid to the *diet*, the coarser vegetables and sweets being prohibited, since they increase the pain by exciting the production of gas. A change of air has improved the condition in several instances occurring in my own practice. The usual constipation may be relieved by simple enemata or by the use internally of the fluid extract of cascara sagrada. Tonics and alteratives, the latter with a view to promoting the absorption of the exudate, may also be employed, and I would recommend especially for this purpose the double iodids, as in the formula given in the discussion of *Pleurisy* (*vide* p. 556). In the early stages some degree of relief, or even a curative effect, may be secured by *local means*, as the application of equal parts of belladonna and iodine ointments until mild counter-irritation is produced. Ichthyol ointment is also serviceable. After all, however, little is to be gained from therapeutic measures, and it is to surgery that we must look for fresh triumphs in the treatment of this truly distressing complaint. Cases of chronic localized peritonitis with adhesions have been operated upon successfully by W. E. Ashton, H. A. Kelly, and others. Instances of chronic generalized peritonitis, whether tuberculous or not, in which the fluid effusion reaccumulates rapidly after repeated tapplings, also furnish adequate indications for operative procedures.

## ASCITES.

(*Hydrops Peritonæi; Dropsy of the Peritoneum.*)

**Definition.**—An accumulation of serum in the peritoneal cavity, resulting from stasis (obstruction) in the branches of the portal vein.

**Pathology.**—The quantity of liquid contained in the peritoneal cavity is quite variable, though it often amounts to several gallons. It is clear and transparent, or slightly opalescent, especially on standing, and the specific gravity ranges from 1010 to 1014. In color it often has a faint lemon-yellow tint; it may, however, be either distinctly yellow, brownish (in cirrhosis), bile-stained (as when jaundice is present), or slightly blood-stained. In reaction it is usually alkaline; very rarely it is either acid or neutral.

The ascitic fluid usually contains much albumin, resembling in this respect blood-serum, as would be expected from its source. The percentage of albumin may be approximately ascertained by noting the specific gravity of the fluid by the urinometer. Thus, in true ascites the specific gravity ranges from 1010 to 1014, and the variation in the percentage of albumin is from 1 to 2. In effusions due to *peritonitis* the percentage of albumin ranges higher (2.5–6 per cent.); hence the specific gravity ranges correspondingly higher (1015–1024). The standing specimen may show to the unaided eye a minute coagulum of fibrin. In the lowest layer of the fluid the microscope discloses leucocytes, red blood-corpuscles (in abundance when ascites is due to general venous stasis), fat-cells, endothelium, and cholesterin-crystals. In ascites the microscopic appearances of the peritoneum are usually normal, while in instances of *peritonitis* the membrane, including the subperitoneal fibrous tissue, is opaque and slightly thickened.

In the so-called *chylous ascites* the fluid resembles milk; it contains fat-droplets, a few lymphocytes, and sugar (Hodlmoser<sup>1</sup>). This condition may be associated with a collection of milky fluid in the left pleural sac, when there is thrombosis of the subclavian vein at the point at which the thoracic duct enters. The term *ascites adiposus* is applied to a milky fluid, in which the origin of the fat is the débris of degenerated epithelial cells, with few fat-droplets and no sugar (Quinke and Senator), to the exclusion of other morphologic elements.

In long-standing cases the abdominal and the thoracic organs become atrophied from pressure exerted by the dropsical fluid. The heart is elevated.

**Etiology.**—Among the chief causal factors are those that hinder or arrest the return of venous blood from the peritoneal membrane, as the following: (a) Pressure upon the branches of the portal vein within the liver, due to contraction of surrounding tissues, as in hepatic cirrhosis (including malarial atrophy—De Brun), syphilis of the liver, and cancerous infiltration. (b) Numerous conditions in the course of which pressure may be made upon the portal vein external to the liver, as enlargement of the glands in the fissure, carcinoma, hydatids, or abscesses connected with the liver. Tumors of any adjacent organs (*e. g.* pancreas) may produce it. (c) Thrombosis of the portal vein. (d)

<sup>1</sup> *Wiener klin. Woch.*, 11 Jahrg., No. 49.

Pressure upon the inferior vena cava after it receives the hepatic trunk (Roberts), or upon the latter itself, or the lymphatics. (e) The portal circulation is also impeded in chronic pulmonary affections (cirrhosis and emphysema) and heart-diseases (e. g. ascites due to "pericarditic pseudocirrhosis of the liver"—Pick). (f) A new growth in the peritoneum may compress the smaller veins lying in the membrane or the root of the mesentery. (g) Diminished resistance of the walls of the portal vessels, due to chronic affections that diminish the albuminous constituents of the blood and impair the nutrition of the peritoneum, as Bright's disease, carcinoma, syphilis, chronic malaria. (h) Chylous ascites is caused either by a leakage of the lacteals (due to ulceration, injuries, or the presence of filariæ) or by the obstruction of the thoracic duct (due to thrombosis, cicatrices, compression). (i) Adipose ascites has for its direct cause fatty cellular degeneration, such as is found in carcinoma, tuberculous and other forms of chronic peritonitis.

Leyden has recently (1897) described an ameboid organism observed in the ascitic fluid in 2 cases.

**Symptoms.**—Slight peritoneal dropsy gives rise neither to symptoms nor to abnormal physical signs. When the sac contains 1 quart (1 liter) of fluid or over, however, the first subjective *symptoms* that are due to the mechanical effect of the fluid appear. They are a sense of weight and fullness, with slight uneasiness. As the proportion of transuded serum becomes gradually increased these symptoms become more pronounced. There may in addition be a *dragging pain* in the loins, *gastro-intestinal disturbance* (meteorism, constipation), and dyspnea (owing to the resistance opposed to the descent of the diaphragm, resulting in compression of the lungs). The latter symptom is much increased upon exertion or on assuming the recumbent posture. Since the *heart* is displaced upward, an embarrassment of its action (rapidity and irregularity) would be expected. *Syncope* is not infrequent for similar reasons. Frequent *micturition* from pressure upon the bladder is common, and the kidneys, owing to compression of the renal vessels, secrete an *albuminous urine*, which is greatly lessened in amount.

**Physical Signs.**—After the serum has collected in considerable amount the physical signs afford characteristic evidence of the condition. From *inspection* we learn many valuable points: (a) The belly is uniformly prominent (the degree depending upon the amount of serum present), giving a rounded form. Changing the posture of the patient shifts the point of greatest pouching. (b) The skin is seen to be tense, smooth, and shining, and sometimes shows lineæ albicantes; the umbilicus commonly bulges forward; less frequently it is obliterated, and the surface-veins are often enlarged. (c) The thorax appears small, except at the base, where it is distended, and the ensiform cartilage is sometimes abruptly curled up. (d) The respirations are hurried and are of the thoracic type, the abdominal movements being slight or entirely wanting. As soon as the belly-walls become moderately tense *fluctuation* is elicited by placing the palm of the left hand vertically upon one side of the abdomen, and then, with the finger-tips of the right hand, tapping lightly the opposite side; impulses thus sent through the fluid will be distinctly felt by the hand in contact with the abdomen. When the dropsical fluid is small in quantity the patient should assume the erect



posture during the examination. In palpating the solid organs (liver, spleen, abdominal tumors) when ascites is present, the tips of the fingers only are placed upon the skin, and then are suddenly "dipped," displacing the fluid, thus touching the solid organ or new growth. *Percussion* gives dullness, even to flatness, over all of that portion of the abdominal cavity occupied by the fluid. The upper level of dullness is not represented by straight transverse lines, but presents a concavity that is pointed to the head. The dullness is extremely movable, shifting as the patient's position is changed. When the decubitus is supine the most dependent portions of the abdomen give dullness. Again, if the patient be made to lie on either side, the opposite or uppermost flank will be found clear, the ascitic fluid always gravitating to the bottom of the sac. Tyson has observed that the flanks are tympanitic with considerable frequency in ascites, and my own experience has been similar, tympany over the head of the colon being almost constant, except in pronounced cases. Moreover, to obtain reliable results, if the layer of fluid be thin, the pleximeter finger is pressed lightly upon the surface, and the gentlest percussion only is allowable. In the cardiac region there is often percussion-resonance as high as the fourth rib, and occasionally a murmur may be heard at the base.

**Diagnosis.**—In order to arrive at a positive diagnosis a clear history of one or the other of the known causative conditions is requisite, joined with distinct evidence of the presence of fluid—viz. fluctuation and movable dullness. For the early diagnosis of ascites the patient should be placed in the knee-elbow position, when dullness can be readily elicited in the umbilical region.

The **diagnosis of chylous ascites** and **ascites adiposus** rests upon insecure ground unless aspiration be resorted to, although the presence of the causative conditions in the case may afford a basis for suspicions.

**Differential Diagnosis.**—Ascites is most apt to be mistaken for an *ovarian cyst*. The accompanying table presents the chief differentiating points:

## ASCITES.

## OVARIAN CYST.

*Clinical History.*

General health is bad prior to the appearance of the enlargement.

General health is good before the development of the tumor; failure afterward.

History of disease of liver, lungs, heart, kidneys, or other organ.

Frequent history of dysmenorrhea, negative as to organic affections.

Swelling begins below and gradually extends higher; more noticeable when sitting than in the standing posture.

Swelling is unilateral at first, gradually becoming more central.

*Physical Signs.*

Enlargement is symmetric, the abdomen being rounded and most prominent about the umbilicus; in the supine posture the abdomen flattens, with lateral bulging; the umbilicus is often pouched and thin.

Enlargement is asymmetric or irregular, unless the tumor be very large, when it may fill the entire abdomen. The greatest circumference is below the umbilicus, which never bulges.

Fluctuation is general from side to side and in a vertical direction.

Fluctuation is circumscribed, corresponding to the limits of the tumor.

## ASCITES.

*Physical Signs.*

No aortic pulsation felt.

Vaginal examination often shows the uterus to be movable. A pouch may project into the vagina, but no cyst is detectable.

When standing, the upper line of dullness is concave.

In the supine position the flanks are especially dull, with tympany in front. Dulness is movable according as the position is altered.

## OVARIAN CYST.

Aortic pulsation is sometimes evident.

Vaginal examination shows the uterus to be displaced. A cyst may be felt and outlined in the pelvis.

When standing, the upper line of dullness is uniform or convex.

In the supine position dullness is still in front and the flanks are resonant.

The area of dullness is not varied by change of posture.

*Character of the Fluid.*

Ascitic fluid has a specific gravity of 1010-1014, and is usually clear. It is of a pale straw-color.

Ovarian fluid has a specific gravity of 1018-1054. It is of a thick, turbid character, and the color is variable.

It should be recollected that large cysts may spring from other abdominal organs than the ovaries, as the *pancreas* and *liver*; the elimination of these latter conditions, however, does not, as a rule, offer marked difficulty. Ascites must be distinguished in practice from the exudation due to *chronic peritonitis*, and the points of differentiation have been arranged thus:

## ASCITES.

A previous history of organic disease of the liver, heart, kidneys, or other organ is obtainable.

No pain is experienced.

The abdomen is symmetrically enlarged.

Fluctuation is general in the transverse or vertical directions.

Palpation detects no hard masses of irregular prominence.

Dulness is always movable upon altering the position of the patient.

The fluid consists of serum with few morphologic elements. It is limpid, with a specific gravity of 1010-1014, and is pale straw-yellow in color, often with a greenish tinge.

## CHRONIC PERITONITIS.

There is a previous history of acute peritonitis, tuberculosis, or inflammatory diseases of the female pelvic organs; sometimes a history of injury.

Pain is a prominent symptom.

Abdomen is irregularly prominent, and rarely flat.

Fluctuation is often limited to circumscribed areas due to loculation of fluid.

Palpation often detects resistant, uneven prominences.

Dulness often not changeable on varying the position, owing to adhesions.

The fluid is either sero-fibrinous, seropurulent, or milky in nature. It is often viscid, and its specific gravity is 1018-1024. The color varies.

*Over-filling of the bladder* has also been confounded with ascites, and this organ has been tapped under the mistaken notion that the condition was one of dropsy of the peritoneum. If, however, the precaution be taken to catheterize the patient before tapping for supposed ascites, the error cannot, as it should not, occur.

**Prognosis.**—The duration of ascites may be for many months or even years. In most instances the prognosis is unfavorable, though modified by the character of the causal condition in individual cases. The immediate cause of death may be either syncope, asphyxia, pulmonary atelectasis from compression of the bases of the lungs by the diaphragm, or it may be the causal disease.

**Treatment.**—**Dietetic.**—The diet should be largely nitrogenous,

light, nutritious, and given at stated periods with a view to maintaining the normal proportion of albuminous material in the blood.

**Medicinal.**—By means of therapeutic measures we should aim to accomplish two things: First, the improvement or cure of the original disease; and secondly, to relieve the chief symptoms by removing the ascitic fluid on which they depend. Though the causative affection is usually chronic and incurable, every effort should be made to remove or mitigate its pernicious activity in accordance with the principles laid down in appropriate portions of this work. Of medicines used to remove the transudation, hydragogue cathartics are most potent for good, and particularly when the ascites is due to cardiac or renal disease. Calomel and jalap in combination, or salines in full doses, administered after the Matthew Hay method, should be tried. Diuretics are also recommended, and English authors greatly praise copaiba and its resin as being among the best. The bitartrate and other salts of potash, either alone or in combination with juniper and digitalis, are of signal value. Sabrazles and Dion have recently recommended urea as an effective diuretic in ascites due to benign forms of atrophic cirrhosis. Equally important with the exhibition of the above remedies is the use of tonics, including hematinics, to promote the general nutrition of the patient. I have reported one instance, occurring at the Philadelphia Hospital, in which a cure was effected perhaps solely as the result of measures intended to assist the nutritive processes. In ascites due to cirrhosis of the liver recourse should be had to *paracentesis abdominis*, not as a last resort only, but “as a systematic method of treatment” (Roberts). A single tapping is rarely sufficient, and a repetition of the measure from time to time, until the collateral circulation is established, is to be advised and encouraged. In cases in which the transuded serum has rapidly re-formed after its removal by tapping, Southey’s tubes, by means of which permanent drainage is secured, have been used with good results. Drummond affirms that ascites due to liver-cirrhosis can be cured, and has proposed an operation whereby adhesions between the abdominal contents and its parietes are secured, in which new blood-vessels are formed, thus establishing an efficient collateral circulation.

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## NEW GROWTHS IN THE PERITONEUM.

THE most frequent and important of the new growths of the peritoneum are (a) carcinoma and (b) tuberculous deposit and tuberculous peritonitis, the latter two having been already considered.

### CARCINOMA OF THE PERITONEUM.

There occur the usual varieties—scirrhous, encephaloid, and colloid—the latter most frequently involving the omentum. Primary carcinoma of the peritoneum is rare. Primary endothelioma, however, is occasionally met with. It resembles true carcinoma in macroscopic as well as in microscopic appearances, though it is in reality to be ranked



with the sarcomata on account of its origin. Carcinoma of the peritoneum is almost always secondary to carcinoma of the stomach, liver, or pelvic organs. The peritoneum may either be the seat of numerous small round miliary tumors, or, less commonly, of larger and distinctly nodular masses, the most extensive development being presented by the colloid variety. Cancerous peritonitis is commonly found to be an associated condition, and the retro-peritoneal lymph-glands not infrequently show cancerous development.

**Etiology.**—More cases occur in the female sex than in the male. Age has also a potent influence, most cases appearing late in life.

**Symptoms.**—When *primary*, carcinoma of the peritoneum is obscure during the early part of its course. Local *pain* and discomfort are complained of, and clinical evidences of the *cancerous cachexia* develop early, but these symptoms are not at first striking enough to be entirely characteristic. Later, however, the *nodules* can often be plainly felt (unless the liquid effusion be too marked), and the *ascites*, *loss of flesh*, *weakness*, and *anemia* are now sufficiently developed for diagnosis. In the colloid variety ascites is often absent, the abdominal cavity being the seat of a large, semi-solid, non-fluctuating mass.

The *secondary form* usually follows carcinoma of the stomach or the ovaries, and the cachexia will have been developed before the peritoneum is secondarily involved in consequence of the presence of the primary growth. Hence, any symptoms referable to the general abdominal cavity are strongly suspicious. Among other *constitutional symptoms*, apart from those already mentioned, is fever (rarely absent), which may be due in small measure to the anemia, though in a greater measure to the associated peritonitis.

**Physical Signs.**—The abdomen protrudes if effusion be present or if the carcinoma be of the colloid form, though this cannot be set down as a uniform rule. Even when the tumor is large, dropsy of the peritoneum sometimes makes its detection impossible. On practising palpation after tapping, however, the nodules can be easily made out, either extending from side to side or being more or less localized and not adherent to underlying structures.

**Differential Diagnosis.**—It will be remembered that an oblong tumor lying in a transverse position below the stomach is met with in certain forms of *chronic peritonitis*. This offers the same physical signs that are presented by cases of peritoneal carcinoma, unless the tumor-masses in the latter affection be of considerable size. Carcinoma, however, is most apt to occur in persons past middle life, while nodular tuberculous peritonitis appears almost exclusively in children and young adults. Evidences of tuberculous disease elsewhere, past or present, and particularly suppuration about the umbilicus, would point to tuberculous peritonitis. Moreover, in all forms of abdominal carcinoma the inguinal glands are apt to be indurated and enlarged. *Proliferative peritonitis* usually gives a history of chronic alcoholism. The differentiation of *hydatid cysts* of the peritoneum from carcinoma depends upon the history of the case, the presence of hydatid fremitus, the finding of the hooklets in the fluid, the less rapid growth of the tumor, and the lessened amount of pain, fever, and cachexia in the latter disease. *Carcinoma of the intestine* may simulate somewhat the disease under

consideration, but the signs of increasing stenosis, as evidenced by the colicky pain, the discharge of blood and pus with the stools, and the ribbon-like character of the feces, will serve to separate the conditions. *Retro-peritoneal tumors* (sarcomata) are discriminated with the greatest difficulty. Tumors of the peritoneum, however, whether of the omentum or mesentery, are movable, while those behind the peritoneum are immovably fixed. Omental tumors lie in front of the intestines (as can be shown by inflation of the bowel); mesenteric new growths sometimes have a coil of intestine in front of them. On the other hand, retro-peritoneal tumors are always crossed by loops of intestine. Peritoneal tumors (particularly the omental) follow the movements of respiration, while the retro-peritoneal remain immobile. The latter always cross to some extent the central long axis of the body, while the former may be confined to one or the other side.

The **prognosis** is always unfavorable.

**Treatment** can accomplish nothing beyond a more or less complete relief from the distressing symptoms.

## PART VII.

# DISEASES OF THE URINARY SYSTEM.

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## I. DISEASES OF THE KIDNEY.

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### MOBILITY OF THE KIDNEY.

(*Movable Kidney; Dislocated Kidney; Floating Kidney; Wandering Kidney; Ren Mobilis; Nephroptosis.*)

**Definition.**—A distinction is made between two common varieties of mobile kidney, according to the degree of displacement, as follows: (1) *Movable kidney*, the upper end of which can be felt during deep inspiration, and which can be pushed down in the retro-peritoneal space to the level of the umbilicus; (2) *Floating kidney*, which is freely movable below or beyond this point—*i. e.* possessing a larger arc of mobility. In the so-called *palpable kidney* the lower edge of the organ can barely be felt on deep pressure.

**Etiology.**—Although an anomalous position of the kidney is usually acquired, it may be congenital; in such cases the condition may be due to relaxation of the perinephric (peritoneal) tissues, the kidney thus having a mesonephron and floating freely about in the abdominal cavity. An abnormally long renal artery may also predispose to the development of a movable kidney.

Emaciation with a marked wasting of the fatty capsule in which the kidney is imbedded is a frequent underlying cause of movable kidney. Women are oftener affected than men, and relaxations from multiple pregnancies, tight-lacing and girdling, and traumatism (falls, heavy lifting, and the like) have frequently caused displacement and mobility of the kidney. Suckling<sup>1</sup> observed that a number of girls who served beer, and were therefore obliged to stoop and immediately stand upright with considerable frequency, were likely to have movable kidney. Heavy tumors of the organ, the pressure of adjacent tumors (as of the liver), and the traction of hernias may likewise cause the condition.

In *enteroptosis*, or Glénard's disease, in which there is a downward displacement of all the viscera, mobility of the kidney is often associated. Although either kidney, or even both kidneys, may be abnormally mobile, the right one is usually affected, probably owing to its anatomic position and to its relation with the liver above. Sometimes a floating kidney becomes fixed by peritoneal adhesions in an abnormal position, as in the right iliac fossa; an instance of this occurred in a seaman, under my care, admitted to the Medico-Chirurgical Hospital

<sup>1</sup> *Edinburgh Med. Journ.*, Sept., 1898.



of Philadelphia. In this case the dislocation of the kidney was apparently caused while on shipboard by a prolonged and intense straining at stool after taking a large dose of castor oil.

**Symptoms.**—Movable kidney may exist without any symptoms whatever. It may be discovered accidentally by physical examination, and not infrequently it is found *postmortem* in a similar manner, no history of trouble having been elicited during life.

The symptoms of movable kidney are local, reflex, and general, the *local* and *reflex symptoms* being the most prominent in the average case. The reflex symptoms, though usually abdominal, may become general. The local symptoms are most marked in extreme mobility of the kidney (floating kidney), while in moderate mobility the reflex symptoms usually predominate over the local.

Most frequently there is a troublesome dragging pain, or a sense of weight or pressure in the loins or abdomen, especially after long walking or standing or hard labor; this may, at times, be referred to the sacral region. Sometimes the pain may be quite sharp and colicky in nature. Pain in the kidney itself is seldom complained of, and then only in those comparatively rare conditions in which congestion is produced by pressure or traction upon the renal veins, obstruction of the ureter, or the like. The patient himself sometimes recognizes the kidney as a tumor, tender and distressing, as in a case under my own care.

*Reflex gastro-intestinal disturbances* are common. Indigestion is usually complained of, and occasionally nausea and vomiting are noted. Dilatation of the stomach may possibly be caused by a dislocated kidney pressing upon the duodenum, but the association of the two conditions is probably coincident rather than causative. Pressure-jaundice is also an unusual concomitant of the floating kidney. Cardiac palpitation, constipation, flatulence, and edema of the lower extremities (from pressure on the inferior vena cava) may attend, and disturbances of the pelvic viscera have also been noted occasionally (dysmenorrhea, abortion, and irritable bladder). Improvement is usually considerable in pregnancy. Some cases of displaced kidney are characterized by sudden and severe attacks of nephralgic or gastralgic pains, chills, fever, vertigo, nausea and vomiting, and general collapse. These attacks are often periodic, occurring sometimes at the menstrual period, and are known as "*Dietl's crises*" or "*incarceration symptoms*." They may be excited, also, by a too free indulgence in eating and drinking, as in a case reported by Osler. It is most probable, as Dietl himself suggested, that these cases are due to a twisting or bending of the renal vessels or of the ureter, or, perhaps, to circumscribed inflammation of the mobile kidney. An acute hydronephrosis may thus develop, with diminished diuresis. The urine is concentrated, and may contain uric acid or oxalates in excess. After three or four days, as the attack subsides, micturition becomes free, the swollen and sensitive kidney becoming movable once more. When produced by movable kidney these attacks of *transitional hydronephrosis* may occur intermittently (*vide* Hydronephrosis).

*Floating kidney* associated with Glénard's disease (enteroptosis, splanchnoptosis), in which the transverse colon, pancreas, stomach, intestines, and other viscera are prolapsed, owing to looseness and weakness of the visceral attachments, gives rise to symptoms similar to

those stated above, only with the addition of greater discomfort and nutritive and nervous disturbances. Sometimes there is albuminuria.

The *general symptoms* of movable kidney are those of "nervousness" neurasthenia, or hysteria. Mental anxiety, leading to melancholia, sometimes follows the discovery by the patient of a movable abdominal tumor, which is persistently believed to be a "cancer." Cephalalgia, backache, mental irritability, paresthesias, neuralgias, nervous dyspepsia, and various hysteric manifestations may arise and prove a perpetual annoyance. In those less frequent instances in which men are affected with mobile kidney hypochondriasis may develop.

The **physical signs** of movable or floating kidney are highly important and diagnostic. *Palpation*, especially bimanual, as by Israel's method. The patient lying in a semi-recumbent position, counter-pressure (the left hand being placed over the lumbar region, the right next the skin in front, manipulating the abdomen from above downward) may detect a firm, movable tumor of renal size and shape in either flank (usually the right) just below the ribs (movable kidney), or in the inguinal or umbilical regions (floating kidney). Though comparatively easy to outline, the tumor is nevertheless hard to grasp; it is often, however, readily pushed into place. Deep breathing may affect a palpable or movable kidney, but has no effect upon one that freely wanders about the abdomen (floating kidney). Pulsation of the renal artery may be felt in the last-named cases.

*Inspection* and *percussion* of the lumbar region in movable kidney are uncertain, and therefore unreliable. Visible depression here is rarer than a visible tumor anteriorly; the latter, however, is not common, although it is occasionally noted in cases of marked wandering of the kidney, as to the inguinal region. I have noted increased tympany over the affected side in several cases as compared with the same area on the opposite side.

A **diagnosis** is possible only after a careful and thorough physical examination. When this is made, an abnormally mobile kidney is usually discovered without difficulty. The size and shape of the organ, its right-sided position, and its mobility, associated with a train of local, reflex, or general nervous disturbances, especially in a thin, emaciated woman, are quite distinctive. A standing (preferably bending forward with the hand resting on a table) or knee-elbow posture is sometimes more favorable than the recumbent position for determining a movable kidney, and a lax abdomen greatly facilitates the physical exploration.

Floating kidney is of course more easily diagnosed than the movable type, and partly because of the fact that in instances of the latter, tumors of the gall-bladder especially, and wandering spleen must first be excluded. The absence of a well-defined splenic notch, the presence of pulsation of the renal artery, a tympanitic note over the usually intervening colon, and an unchanged area of splenic dulness will assist in the diagnosis; in addition there is the fact that wandering spleen is a comparatively rare affection.

**Differential Diagnosis.**—Tumors of the gall-bladder, as Henry Morris has shown, are frequently mistaken for movable kidney; occasionally the opposite error is made; sometimes both conditions may exist. They are both common to women; the right kidney is more often movable than the left; they both may present as tumors in the right hypochon-

driac and umbilical regions; they are more or less movable, firm, smooth, slightly tender, round or oval in shape, with variable percussion-signs, and dyspeptic symptoms; and either may give rise to paroxysms of severe colic, or to jaundice. Jaundice, however, is probably rare in movable kidney, while emaciation and general nervous disorders are more common; the floating tumor is also less easily palpated than the cholecystic, and may vary in size (hydronephrosis), the diminution being accompanied by a marked increase in the flow of urine. If the gall-bladder be filled with calculi, the consistence is firmer than that of the kidney, and fremitus may be felt. Moreover, the movements of the gall-bladder are usually lateral within a short arc of a circle, the center of which is a point beneath the edge of the right lobe of the liver; while those of floating or movable kidney may be either vertical, oblique, or lateral in arcs of a much larger radius. Again, tumors of the gall-bladder descend with inspiration, as is not the case with wandering kidney.

In some cases it may be necessary to distinguish between the attacks of pain known as "Dietl's crises" and renal, hepatic, or intestinal colic, acute intestinal obstruction, and appendicitis; the symptoms peculiar to these conditions must then be considered in forming a diagnosis.

Tumors of the ovaries and bowel are rarely confounded with wandering kidney.

**Prognosis.**—In uncomplicated cases life is never endangered, and a cure may be effected in a large majority of cases in which suitable combined medical and surgical treatment is pursued. The general nervous symptoms are usually very obstinate, but after relief is afforded from the local and reflex symptoms, whether by tentative or operative means, they subside or cease altogether.

**Treatment.**—Since emaciation and loss of perirenal fat is a frequent cause of wandering kidney, it is often advisable to resort to measures that will tend to increase the weight and fat of the body. The "rest-cure," with its forced feeding, may be all that is necessary in highly nervous subjects having but a slightly movable kidney. In all cases more or less prolonged intervals of rest (lying down) throughout the day aid markedly in ameliorating the symptoms. Other hygienic measures, as the avoidance of over-exertion, extreme bodily movements, straining—as at stool—and so forth, should also be enjoined.

For several years, and until recently, the operation for anchoring the mobile kidney has been advised as appropriate in nearly all cases. This is now perhaps wisely deprecated; and a reversion to the careful, patient, and constant use of suitable abdominal pads and binders in certain cases is meeting with much success in affording comfort and support, besides a marked reduction in the reflex, abdominal, and general nervous symptoms. In severe cases of renal displacements, in which recurring attacks of hydronephrosis, strangulation-crises, profound nervous and mental disturbances, or other grave renal complications occur, some such surgical procedure as nephrorrhaphy may be necessary. This often proves an effectual cure, although occasionally the anchorage may be torn loose by a sudden or severe physical effort. Total extirpation of the kidney (nephrectomy) is justifiable only in the gravest cases and after other means have failed. The hypodermic injection of morphin and atropin and the external application of heat are indicated in the crises of Dietl.



## CIRCULATORY DISORDERS OF THE KIDNEYS.

## ACTIVE HYPEREMIA.

*(Acute or Active Congestion.)*

**Definition.**—An acute, temporary engorgement of the vessels of the kidneys, with little or no exudation.

**Pathology.**—The kidney is swollen, deep-red in color, and engorged with blood, which flows freely on section. Microscopically, in severe congestion there may be seen cloudy swelling of the cortical epithelium.

**Etiology.**—Acute renal congestion is due mainly to the action of irritants present in the circulation, as in the acute infectious (especially the eruptive) fevers. The stimulating diuretics and certain poisonous drugs, as copaiba, squills, cantharides, potassium chlorate, and carbolic acid, also sudden contraction of the peripheral blood-vessels by exposure to cold while the body is overheated, act as causes. When prolonged the congestion passes into an acute nephritis. It may be caused in one kidney as a result of the nephrectomy of its fellow. Certain ill-defined centric and peripheral nervous influences and neuroses are held by some to cause an active hyperemia of the kidneys through a vasomotor paralysis of the renal arteries.

**Symptoms.**—There may be a dull pain in the lumbar region, with a slight elevation of the temperature and pulse-rate. The *urine* either is scanty, or, as in cantharides-poisoning, it may be altogether suppressed. It is dark, the specific gravity is increased, and it contains some free blood, a trace of albumin, and a few hyaline tube-casts.

**Diagnosis.**—The absence of a marked quantity of albumin, of the numerous and various casts, of dropsy, and of uremic symptoms distinguishes active hyperemia from *acute nephritis*.

The **prognosis** is quite favorable upon the removal of the cause. It must be borne in mind that a frequent repetition of the attacks may lead to a nephritis.

**Treatment.**—Absolute rest and a liquid diet should be ordered. Cupping over the loins or the use of hot fomentations should be practised. The free use of water and other diluents or mucilaginous drinks should be encouraged. Saline laxatives to freely open the bowels, and the use of hot air or a hot pack to promote sweating, are important aids in relieving the congested kidneys.

## PASSIVE HYPEREMIA.

*(Chronic or Passive Congestion.)*

**Definition.**—A chronic venous engorgement of the renal vessels, generally secondary to diseases of certain other viscera.

**Pathology.**—There is in the later stages a characteristic condition of the kidneys called “cyanotic induration.” Earlier in the case the organs are enlarged, firm, and of a dark, bluish-red color. The capsule is usually non-adherent. On section the medullary substance is seen to be darker red than the cortex and coarsely fibrous in appearance. Micro-

scopic examination shows the capillaries (both glomerular and medullary) somewhat dilated and the walls thickened. The epithelium may either be unchanged or a little cloudy and swollen, or, later, even fatty; the interstitial tissue may be slightly increased, especially beneath the capsule of the kidney.

**Etiology.**—Most commonly the renal congestion is a part of a general venous engorgement due to chronic cardiac, pulmonary, or hepatic disease. It is found in mitral valvular disease with ruptured compensation of the heart; in pulmonary emphysema, fibroid phthisis, and chronic adhesive pleurisy; and in cirrhosis of the liver. The “*cardiac kidney*” is the commonest variety. Less frequent causes of congested kidneys are tumors, the pregnant uterus, and ascites, all of which bring about the condition through pressure upon the renal veins. Only rarely may passive renal congestion be due to thrombosis of the ascending vena cava or of the renal veins.

**Symptoms.**—These are accompanied by those due to the primary diseases that are manifested in the general venous congestion, as *edema* of the lower extremities. There may be a sensation of weight in the loins. The *urine* is diminished in quantity, of a higher specific gravity, and darker in color; it contains a little albumin, some blood-corpuscles, and a few hyaline casts and epithelial cells, depending upon the chronicity and intensity of the congestion. Urates may be deposited in the standing urine.

**Diagnosis.**—From *nephritis* passive renal congestion may be differentiated by the comparative absence of albumin, casts, general dropsy, and uremia, and by the undiminished quantity of urea.

**Prognosis.**—This depends upon the primary cause. Chronic congestion may pass into chronic nephritis.

**Treatment.**—Rest and a light and easily assimilable diet, together with cardiac tonics and diuretics, are indicated. The infusion of digitalis serves a good purpose by increasing the quantity of urine and clearing it of albumin. Basham’s mixture is a useful adjuvant.

#### EMBOLIC INFARCTIONS.

Anemic and hemorrhagic infarctions of the kidney are of pathologic rather than of clinical significance. Cicatrices may result from these infarctions, giving rise to the “embolic contracted kidney.” Very rarely the *sudden appearance* of a slight amount of *blood* in the *urine*, associated with cardiac disease and possibly with a sudden severe pain over the loin, may point to hemorrhagic infarction.

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### SPECIAL PATHOLOGIC STATES OF THE URINE.

#### HEMATURIA.

**Definition.**—The presence of blood in the urine.

**Etiology.**—(1) *Local* or *renal* causes of hematuria include congestion (including that due to torsion of the renal vessels in certain cases of floating kidney), acute inflammation of the kidneys, and acute ex-

acerbations of chronic nephritis, embolic hemorrhagic infarction, renal calculi and pyelitis, tuberculosis, malignant renal disease, hydatids, traumatism, and parasites (the *filaria sanguinis hominis* and *distoma hæmatobium* (Bilharz)).

(2) *Affections of the Urinary Tract.*—In the ureter, calculi or lacerations due to traumatism, as in protracted and complicated abdominal sections; in the bladder, calculi, malignant tumors, acute cystitis, ulceration and rupture of varicose veins at the vesical neck; and in the urethra, gonorrhea, calculi, parasites, and traumatism,—may all cause hematuria.

(3) *General Diseases.*—Acute specific fevers and certain blood-dyscrasiæ (purpura, scurvy, hemophilia, malaria, and leukemia) may produce hematuria. Malarial hematuria in mild form is not an uncommon feature of paludism in the Middle States of this country, and may occur after the manner of intermittent malarial paroxysms. That due to the renal congestion of chronic heart-, lung-, or liver-disease is not a marked condition, and has not been of frequent occurrence in my experience.

Senator describes an interesting and unusual form of hematuria that is sometimes seen in young persons whose health may be quite fair, the blood often appearing paroxysmally and without apparent cause ("renal hemophilia"). Hematuria may be also a manifestation of vicarious menstruation; and of mountain-sickness (Dieulafoy).

*Endemic hematuria*, so called, is that variety found in some of the tropical regions where the *distoma hæmatobium* (a trematode worm) abounds.

**Diagnosis.**—This has for its object the discovery (1) of blood in the urine, and (2) of the source of the hemorrhage. Bloody urine varies in color according to the quantity of blood present, to its condition (coagulability), disposition, and the length of time present in the urine. A light reddish tinge may indicate a slight quantity of blood. A dark coagulum may be at the bottom as a sediment, with small clots floating above in a deep-red, turbid layer, above which, again, the urine may show but the slightest tint of red. Or the urine may have a smoky-red or chocolate-hued appearance. Microscopically, the blood-corpuscles are readily discovered, establishing the diagnosis from hemoglobinuria, in which condition they are absent. When red corpuscles are associated with tube-casts, renal hemorrhage may be positively diagnosed. In ammoniacal urine or in urine of low specific gravity the corpuscles are very pale and shadowy (dissolved hemoglobin). After remaining in ordinarily acid and diluted urine they lose their disk-like shape and swell into spheres of a smaller diameter. Urine containing blood always shows the presence of albumin. According to Newman,<sup>1</sup> a ratio of albumin to hemoglobin in excess of 1 to 1.6 indicates not only an independent albuminuria, but also a renal affection as the cause of the hematuria.

*Chemically*, the blood-pigment may be detected by Heller's test, which consists in adding liquor potassæ, boiling the urine, and observing the flakes of precipitating phosphates, which become reddish-yellow or brown from the added hemochromogen. The guaiacum test is also used. The spectroscope is sometimes employed to discover the bands produced by the blood coloring-matter.

The source of the blood in hematuria is of great diagnostic and

<sup>1</sup> *Lancet*, July 9, 1898.



therapeutic importance. In *renal hemorrhage* the blood is thoroughly mixed with the urine, giving a uniformly red or brown color, as in hemorrhagic nephritis. Blood-casts and leukocytes may also be found. The disease causing hematuria may be traced sometimes by a study of the urine; thus, in cases of valvular cardiac disease the sudden appearance of hematuria would indicate *infarction* of the kidney. The discovery of a few red blood-corpuscles in a concentrated urine would point to renal congestion. In profuse renal hemorrhages clots representing moulds of the renal pelvis and of the ureters may be discharged. Hemorrhage due to *calculus* is usually small in amount and appears at more or less prolonged intervals. Tubercular hemorrhages may occur very seldom.

Blood from the ureters is usually moulded in clots in the shape of curved cylinders, and appears like small dark worms in the urine. Casts from the ureters are often secondary to hemorrhages; in such cases the hematuria may alternate with the passage of clear urine, owing to temporary hemorrhages or to the blocking of the ureter on the diseased side. (See also Fibrinuria.)

*Vesical hemorrhages* may be quite copious. The blood and urine are not intimately mixed, and large clots settle on standing. The first portions of urine discharged may not be bloody, while the last portion may consist of pure blood.

Finally, *urethral* blood is discharged before the urine, and either comes away freely or may be "milked out" independently of urination.

The endoscope has been used successfully to determine the source of the hemorrhage. It is especially useful in women. It is also possible with this instrument to determine which kidney is affected.

**Prognosis.**—This varies with the primary source of the hematuria.

The **treatment** consists primarily in rest in bed. The application of dry cold to the loins is useful, and the hypodermic injection of ergotin is to be recommended for trial. Internally, such hemostatics as the extract of hamamelis virginica, the extract of hydrastis canadensis, gallic acid, lead acetate, ergot, and opium may be used. Cantharides tincture in 2 to 5 drop doses has been tried with good results in hematuria due to renal congestion.

#### HEMOGLOBINURIA.

**Definition.**—The presence of blood-pigments, especially methemoglobin, in the urine.

**Etiology.**—The direct cause of hemoglobinuria is a condition of the blood in which, as a result of the dissolution of the red corpuscles, the hemoglobin is set free and is excreted by the kidneys.

(1) The causes of the hemolysis are principally *toxic*, and include the following: (a) Poisons (carbolic and pyrogallic acids, potassium chlorate, naphthol, phosphorus, arseniuretted hydrogen, and carbon dioxid). (b) The ingestion of poisonous fungi or of tainted edible mushrooms (*Helvella æsculenta*). (c) The poisons of certain infectious diseases (scarlatina, typhus and typhoid fevers, yellow fever, syphilis, scurvy, purpura). (d) Extensive burns, the absorption of hemorrhagic effusions, and the transfusion of animal blood. (e) Rarely it may be due to exposure to cold and to violent physical exertion. (f) Finally, there is the so-called *epidemic hemoglobinuria* (Winckel's disease) that occurs in the new-born.

(2) **Paroxysmal hemoglobinuria**, a rare and interesting variety, may occur without any apparent cause in persons enjoying otherwise good health. It appears thus distinctly as an independent disease. Some of these cases, however, have been attributed to a peculiar susceptibility to cold (generally or locally applied) and to marked exertion; especially is this the case in adult white males. It is held by some to be a manifestation of Raynaud's disease, and by others to be the result of syphilis.

(3) It appears as a symptom of *malaria* (which acts like the other infectious diseases) in the southern part of this country, where the pernicious varieties of malarial toxemia are most common. This is termed *malignant malarial hemoglobinuria*. In Africa it is called *black-water fever*.

**Symptoms.**—These are generally the symptoms of the condition that accompanies hemoglobinuria. In paroxysmal hemoglobinuria the attacks are usually sudden, brief in duration, and sometimes *intermittent*, especially when of malarial origin. An anemic condition seems to be essential to the production of malarial hemoglobinuria. *Jaundice* may be an associated symptom. The hemoglobinuria seldom lasts for more than two days, though very grave cases take on the aspect of a pernicious malarial attack. There may be lumbar pains, chills and fever, and gastric disturbances. Urticaria and purpura have also been noted, as has anemia in cases in which frequent attacks have taken place.

**Diagnosis.**—This is made by an examination of the urine. Macroscopically, it is of a red-brown color, slightly turbid, with a reddish-brown or brownish-black sediment. The reaction is usually acid, and the specific gravity slightly lowered. The microscopic features that distinguish hemoglobinuria from hematuria are variable. In the former condition few or no red corpuscles are present, and the few that may be seen are usually colorless ("shadows") or fragmentary. Small flakes or granules of disintegrated hemoglobin are found, and are brownish-black in color. There may be also brown-tinged casts and epithelium. Chemically, the urine is found to contain albumin, for the discovery of which Heller's and the guaiac tests for blood-pigment may be tried. The former has been described in the preceding discussion of Hematuria. The *guaiac test* consists in overlaying with urine a mixture of the tincture of guaiac and hydrogen peroxid or the oil of turpentine (equal parts). When the blood-coloring matter is present, an indigo-blue ring is formed above a white resinous deposit. When shaken a lighter blue color develops throughout the contents. By means of the spectroscope the three absorption-bands of methemoglobin may be seen (red, green, and yellow). The blood-serum in hemoglobinuria may be somewhat red-tinged on account of the dissolved hemoglobin. The hemoglobinuria is further marked by the aplasticity of the red corpuscles, by their pallor, by poikilocytosis, and by the presence of the irregular flakes of hemoglobin.

The **prognosis** of hemoglobinuria depends upon the cause. It is favorable in the ordinary paroxysmal form. Malignant malarial hemoglobinuria, however, is often fatal.

**Treatment.**—Hemoglobinuria is rather intractable. During the paroxysms external warmth is needed, along with hot drinks to encourage perspiration. In malarial cases quinin, and in syphilitic the iodids, should be administered.

## ALBUMINURIA.

**Definition.**—The presence of albumin in the urine.

**Pathology and Etiology.**—The immediate cause is the escape of the normal blood-constituents, serum-albumin and serum-globulin, from the vessels into the renal tubules. This transudation of albumin indicates either a transient and slight or a permanent and grave nutritional disturbance of either the epithelium lining the glomeruli or of that of the contained tufts of capillaries, or, possibly, of the *membrana propria* or the epithelium of the uriniferous tubules. These changes induce and offer an abnormal perviousness to the albumin of the blood.

The principal *causes* of albuminuria are—(1) Those associated with definite lesions of the kidney; nephritis, acute and chronic; renal congestions, active and passive (the latter being secondary to chronic liver-, heart-, and lung-disease, pregnancy, or tumors); and certain *toxemias*. Among the last-named are included scarlet fever (scarlatinal nephritis) and gout. Other causes are—amyloid and fatty degeneration of the kidney, suppurative nephritis, and renal tumors (cystic kidney).

Albuminuria occurs also in conditions in which (2) the renal lesions are either slight or undemonstrable: (a) Thus, it is present in blood-changes, as in chronic lead-, mercury-, and arsenic-poisoning, scurvy, purpura, syphilis, leukemia, or extreme anemia, and in cases in which urobilin or bile-pigment and sugar (glucose) circulate in the blood. Again, slight albuminuria may be present in pregnancy (*kidney of pregnancy*), in saccharin diabetes, and after etherization. In certain affections of the *nervous system* albumin is found in small quantity, as after an epileptic paroxysm, in tetanus, injuries to the head, apoplexy, and exophthalmic goiter.

(b) The so-called *accidental* or *spurious* albuminuria is due to the presence of pus or blood; in such cases the condition is not a true renal albuminuria, since it is commonly associated with cystitis, pyelitis, urethritis, or is the result of hemorrhage from the pelvis of the kidney, from the ureters, bladder, or urethra.

(c) *Febrile* albuminuria is of rather frequent occurrence in diseases accompanied by pyrexia, especially when long continued. Among these are typhoid fever, small-pox, yellow fever, diphtheria, and even influenza, follicular tonsillitis, and pneumonitis. The renal changes in these cases are, I believe, merely a transitory cloudy swelling in the glomeruli, which, together with the albuminuria, rarely lasts longer than the fever.

(d) Other forms of albuminuria have been styled *physiologic* or *functional*, *transient*, *dietetic*, *intermittent*, and *cyclic*: in these no definite lesions of the kidney are found, and are denied by some to exist. Recent observers are inclined to believe that trivial, non-progressive renal changes occur in these cases. Slight albuminuria certainly does occur in some cases after a heavy meal rich in albumin, after marked and prolonged muscular exertion, intense emotion, and cold bathing.

(e) *Cyclic albuminuria* has come to be of greater interest and importance in later years, particularly as it bears upon the prognosis and upon life-insurance risks. In this variety there is a periodic appearance and absence of albumin in the urine. The albuminuric paroxysms are very variable, recurring usually after meals or on exertion, but generally being absent during rest at night or early in the morning. The albu-



min is present in but small quantity, and only rarely are casts (hyaline) found. The urinary features are otherwise normal, and the accompanying signs and symptoms common to nephritis are absent. Cyclic albuminuria is most common in adolescent anemic males, of poor nutrition (gastrointestinal autointoxication?), dyspeptic, neuralgic, often neurotic, and even hysteric. Under careful management these cases ordinarily recover. There is, however, a class of cases in which the albuminuria is persistent, though but a mere trace of albumin may be detected, and neither can tube-casts be found nor are symptoms complained of. However, an insidious degeneration of kidney-structure may manifest itself many years later, an urgent condition sometimes developing. According to Ranault, albuminuria may rarely be even *hereditary*. Slight *senile albuminuria* in the very old, without any particular evidence of renal disease, is not uncommon.

**Diagnosis.**—This rests upon the discovery of albumin by means of any one or more of the reliable tests shortly to be described. Since albuminuria is no more synonymous with nephritis than is glycosuria or polyuria with diabetes mellitus, it becomes important and necessary, both for prognostic and therapeutic reasons, to differentiate between the so-called functional albuminuria, the cyclic variety, and those associated with coarse and definite anatomic lesions of the kidney.

**Differential Diagnosis.**—Inquiry and careful inference concerning the etiology of a given case must be made. *Renal albuminuria* is persistent and of considerable quantity, except in chronic interstitial nephritis. Tube-casts are usually present. *Functional albuminuria* is slight and inconstant. Tube-casts are either absent or exceedingly few in number in the latter. Again, in the former variety general symptoms, as dropsy, cardiac hypertrophy, anemia, and uremic prodromes, are present. It is true that slight edema is sometimes found in cyclic albuminuria, but this is probably due to the marked anemia that is so often seen. When the quantity of albumin is disproportionately large in *spurious albuminuria*, a suspicion of coexisting renal albuminuria should be aroused (Strümpell).

**Tests for Albumin.**—Two samples of urine, one of the morning before any food is taken, and one of the evening before the patient retires, should be examined. Care should be taken that there be no contamination of the urine with the menstrual, leukorrheal, or urethral discharges. The smallest quantity can be detected only by its coagulum rendering the urine turbid; hence any turbidity present before the given test is made should be removed by filtration, unless this turbidity be due to urates, when a little warming of the tube will render the urine clear.

(1) **Boiling Test.**—This is the commonest and I think the most reliable practical test for albumin. The tube is filled about two-thirds full of urine. If alkaline or neutral in reaction, a drop of acetic or nitric acid is added; an excess of acid must be carefully avoided, lest the albumin (if present) be converted into a non-coagulable form. The tube, held aslant, is then applied to the flame, and slowly revolved with the fingers, so that the upper portion of the column of urine is brought to the boiling-point. A comparison of this with the lower portion of the urine is made. Any turbidity is due to albumin or phosphates. If albumin, adding a few drops of nitric acid will increase and thicken the coagulum; if phosphate, the opaqueness will be cleared at once.

(2) *Heller's Nitric-acid Test*.—This is easily performed, and is both delicate and satisfactory. About 1 c.cm. of nitric acid is poured into a tube, and some urine is allowed to flow slowly from a pipet and settle upon the acid. The presence of albumin is indicated by a white ring at the point of contact of the two liquids. Uric acid, urates, and certain urinary coloring-matters form a pink or deep-red ring or zone; this forms, as a rule, above the juncture of the acid and urine. Hemialbumose also gives a white zone, but does not respond to the boiling test as does serum-albumin.

(3) *Johnson's Picric-acid Test*.—To filtered urine in a test-tube are slowly added a few drops of a saturated watery solution of picric acid. Immediate turbidity indicates albumin. Some authorities prefer that a dram or two (4.0–8.0) of the yellow fluid be placed gently on the surface of the urine, when, if albumin is present, a white zone at once is apparent, together with a haziness that spreads downward with the diffusion of the liquids. Heating emphasizes the evidence of the test, which is extremely sensitive.

(4) *Roberts' nitric-magnesium test* is also very delicate. It consists in using the following mixture, just as in Heller's test: one volume of concentrated nitric acid, added to five volumes of a saturated solution of magnesium sulphate.

(5) *Trichloroacetic-acid Test*.—This will discover minute traces of albumin, but has the disadvantage that it responds to nucleo-albumin as well as to serum-albumin. A few crystals may be dropped into the urine, or a saturated solution may be used after the "contact method," when, if albumin be present, a white coagulum forms. This and the Geisler test-papers (Vierordt) constitute portable and handy tests.

(6) The *acetic-acid and potassium-ferrocyanid test* is also valuable and minutely sensitive. The urine is first made decidedly acid with acetic acid. A few drops of a freshly prepared solution of potassium ferrocyanid are then added, and if either albumin or hemialbumose be present, it will be precipitated.

(7) *Quantitative Test—Esbach's Albuminometer*.—This consists in using a graduated test-tube, into which definite amounts of urine and a reagent composed of 10 parts of picric acid, 20 of citric acid, and enough water to make 1000 parts are carefully mixed by reversing several times the stoppered tube. After allowing this to stand about twenty-four hours, the height of the precipitated albumin is read off on an etched scale, which will indicate approximately the parts per thousand. Not less than 0.5 parts per thousand can be estimated correctly, however. Should there be a hematuria, if the percentage of albumin by Esbach's method, divided into the number of red cells per cubic centimeter of urine, is less than 30,000, it suggests a purely hematuric albuminuria; if greater, it suggests an independent albuminuria (Goldberg).

**Prognosis.**—Etiologic considerations bear heavily in this matter. Functional abuminuria is of favorable import, as a rule. The febrile, hemic, cyclic, and paroxysmal varieties usually clear up with convalescence and with advancing years (in the latter case). The persistence of albumin in these cases, however, even in slight amounts or at variable periods, should cause suspicion, since there must be some glomerular renal change to account for the disorder, the tendency of which is to progress steadily and insidiously. Especially is this true when there

is associated a gradually increasing arterial tension. The presence of tub-casts is conclusive of structural change in the kidneys, marked by degenerations and by exudative and productive inflammation, either acute or chronic.

#### PEPTONURIA AND ALBUMOSURIA.

True peptone (Kühne) has never been demonstrated in the urine. The so-called peptones discovered by Devoto's and other methods are really *albumoses*, and the term *albumosuria* should be substituted for *peptonuria*. These albumoses (proto-, deuterio-, hemi-) are found in acute suppurations or when resolution of inflammatory effusions is going on, as in lobar pneumonia. They may be found also in acute rheumatism, scorbutus, myxedema (Fitz), and in certain forms of metallic and ptomain or bacterial poisoning. Albumosuria may be suspected when, after negative results with the boiling and nitric-acid tests, cold acetic acid produces a cloudiness. This suspicion may be confirmed by the *biuret* test, as follows: Any albumin that is present must first be coagulated and removed. Then, after placing some Fehling's solution in a test-tube, an equal quantity of urine is allowed to come in contact with it, when, if albumoses be in the latter, a rose-pink zone or halo appears at or near the point of contact.

*Hemialbumose* is formed in the urine in osteomalacia, in chronic suppurations, and in sarcomatous disease of the spinal cord, though the clinical or diagnostic significance of the substance has not been fully determined as yet.

#### INDICANURIA.

**Definition.**—The presence of a pathologic quantity of indican in the urine.

Indican occurs in the urine in health in very small quantities as a colorless compound, and is, chemically speaking, indoxyl-potassium sulphate.

**Pathology and Etiology.**—Indican is increased abnormally in the urine by any disorder whereby large quantities of albuminous matters are decomposed. Thus, it occurs in intestinal obstruction, especially when the caliber of the small bowel is diminished from any cause so as to produce a stagnation of the contents and a consequent decomposition from bacterial action, especially the action of large numbers of the common colon-bacillus. Under such circumstances indol and phenol are formed. The former, being absorbed and oxidized into indoxyl, finally appears in the urine in combination with potassium sulphate. Acute peritonitis, obstinate and chronic constipation, wasting diseases, and cachectic conditions in which there is a considerable destruction of albuminoids (as in Addison's disease, neoplasms, cholera Asiatica, and empyema) usually have an associated indicanuria. An increase of the aromatic sulphates in general, or an increase in proportion to the fixed sulphates, is especially significant of intestinal putrefactive processes. Since the pancreatic secretion peptonizes the proteids from which arise leucin and tyrosin, and these in turn are decomposed into skatol, indol, and phenol, it is stated (Piseuti) that any obstruction preventing the flow of the pancreatic juice into the bowel



would be reflected in a diminished quantity of indican in the urine. On the other hand, any epigastric tumor suspected of pressing upon the small intestines would be accompanied with indicanuria.

**Diagnosis.**—This depends upon the demonstration of indican by adding strong oxidizing agents, which decompose this product and set the indigo or pigment free. At times sufficient oxidation of the indican has taken place in the urine before any chemical test is applied, so that a bluish tinge is given thereto. This may be seen in urine that has been standing for some time, the sediment giving a bluish reflection, or there may be a blue-turbid film on the surface. In the urine of indicanuria, moreover, where putrefaction is marked, a pronounced blue-black color may be present.

**Tests.**—*Jaffe's* well-known test consists in mixing equal volumes of urine and hydrochloric acid, and then adding, drop by drop, a concentrated solution of chlorinated lime, shaking the tube after each addition. A strong indigo-blue color appears if there is much indican.

A good modified test is the use of fuming nitro-hydrochloric acid and urine (equal parts) and a saturated solution of chlorinated potash, used as in the above method. A blue-black cloud or ring appears below the surface. If a few drops of chloroform are then added and the mixture is agitated slightly, a blue color settles at the bottom, owing to the chloroform carrying with it the oxidized indican.

#### PYURIA.

**Definition.**—The presence of pus in the urine.

**Etiology.**—Pyuria is due to (1) suppurative inflammation along some portion of the genito-urinary tract, or (2) to the rupture of adjacent abscesses into the tract. According to the source of the pus the urinary manifestations differ in a more or less characteristic manner.

**Pyelitis and Pyelo-nephritis.**—Pus from the pelvis of the kidney may be due to calculous, tuberculous, or other irritation. It is associated at times with the "railed" or transitional epithelium usually seen early in the case. In pyelo-nephritis casts may indicate renal involvement, although it should be borne in mind that in abscess of the kidney pus may be discharged continuously without the appearance of any casts in the urine whatsoever. One such case came to necropsy under the observation of H. S. Anders, in which small uratic calculi were discharged now and then for several years. Later, several larger stones were removed from the bladder by Willard by suprapubic cystotomy, in the hope that by drainage and irrigation of the bladder the marked pyuria might subside or cease. The abdominal opening healed in a few months, and, whilst bladder-symptoms were absent after removal of the calculi, pyuria persisted. Death having occurred suddenly from coronary-artery disease and interstitial myocarditis, it was found *postmortem* that a large abscess occupied the lower third of the left kidney, which was filled with small, dark, and irregularly-shaped calculi. A thick pyogenic membrane surrounded the purulent and calculous contents. No casts were found at any time during life, though repeated examinations were made, and, remarkable as it seems, renal symptoms were altogether absent.

The pyuria is sometimes *intermittent*, one ureter becoming temporarily occluded (on the side of the disease), the clear, normal urine from the healthy kidney passing until the ureteral obstruction is relieved, when pus again appears. Purulent urine from the kidney is usually acid in reaction, except when the pyelo-nephritis is secondary to cystitis, when it is more apt to be alkaline and to contain a decided quantity of mucus. *Cystitis*.—Pyuria in this affection is fetid in most cases. Bladder-symptoms are marked. The urine is alkaline, and a stringy, tenacious muco-pus comes with the last portions. Triple phosphates are often found. The pus and urine are not so intimately mixed as in pyelonephritis.

*Urethritis*.—The pus is in small quantities, is passed in advance of the urine, and can be "milked out" from the male urethra. There is usually a history of gonorrhoeal infection, and the gonococcus may be demonstrated in most cases.

*Rupture of contiguous abscesses* into the urinary tract is accompanied usually with a sudden discharge of a large quantity of pus in the urine, preceded by symptoms of abscess elsewhere, as in the pelvis or right iliac fossa (suppurative appendicitis) or perinephric abscess. The pyuria disappears as abruptly as it came on, or lasts but a few days, lessening gradually until there is a complete cessation. The *strongylus gigas* in the pelvis of the kidney causes pyuria as well as hematuria.

**Diagnosis.**—Pus gives a greenish-yellow or yellowish-white tinge to the urine and sediment, the latter very often becoming very tenacious or jelly-like from the presence of mucus. It may resemble a phosphatic precipitate, as in cystitis; the latter, however, is white, lighter, more granular, and not so thick or tenacious. Microscopically, a positive diagnosis is made by the discovery of pus-corpuscles (or leukocytes) with their granular protoplasm, which has the faculty of clearing up and showing one or more nuclei upon the addition of acetic acid. The corpuscles are either more or less swollen and clear, or opaque, granular, or even nucleated, according to their number, the length of time in the urine, and the degree of alkalinity or acidity of the latter. The greater the change in the urine, the more marked the change in the corpuscles. A few phosphatic crystals may be seen, and epithelium more or less characteristic of the seat of suppuration is present.

Chemically, there is slight albuminuria, a marked amount of albumin usually indicating renal disease. Nephritis may be diagnosed in connection with pyuria by the discovery of casts. On the addition of liquor potassæ to urine containing pus the latter is converted into a clear gelatinoid substance; mucus, on the other hand, becomes thin and flocculent. Mucus may also be distinguished from pus by its failure to react to cold nitric acid, whilst the albumin of purulent fluid coagulates.

#### CHYLURIA.

**Definition.**—The presence of chyle in the urine.

**Etiology.**—This interesting condition may be either *parasitic* or *non-parasitic* in origin. The former type is more common in the tropics, and is caused by an engorgement and rupture of the bladder or renal lymph-vessels, due to obstruction of the larger branches of the thoracic duct or in the duct itself, by the *filaria sanguinis hominis* (*vide* Filaria-

sis). The latter form, the pathology of which is not definitely known, is occasionally found in temperate regions. It is held to follow injuries to the lymphatic ducts, and may be associated with pregnancy.

**Diagnosis.**—The urine is increased in quantity, and has a milky turbidity (*galacturia*) due to the emulsified fat. After standing for a time a light coagulum settles to the bottom and a creamy pellicle of fat rises to the surface. The sediment contains also the fibrin of the chyle. Sometimes as much as 2 or 3 per cent. of fat is present (*lipuria*); this may be tested by agitating a portion of the urine with ether, whereupon the turbidity disappears. Owing to the serum-albumin in the chyle, the various tests for that substance would show traces of its presence in chyluria. Hematuria may be associated with chyluria, especially in parasitic cases, in which case the blood comes from ruptured veins and tinges the urine accordingly. Microscopically, chyle-containing urine resembles milk in its millions of fine granules and fat-droplets.

**Prognosis.**—Chyluria is intermittent in its appearance, corresponding to the times of rupture of the vesical lymphatics, and may last for years. The prognosis of non-parasitic chyluria is good as to life, but unfavorable as to cure.

#### CHOLURIA.

**Definition.**—The presence of bile-pigment in the urine.

**Etiology.**—Choluria may be caused by any disease, local or general, in which *jaundice* is a symptom.

**Diagnosis.**—Bile-stained urine has a color varying from a greenish-yellow to a brownish-green or brown-black, resembling porter. When shaken its foam assumes a characteristic yellow or greenish-yellow color. White filter-paper dipped in the urine is stained yellow.

**Tests.**—The *chloroform* test consists in adding this substance to the urine and allowing it to settle to the bottom of the tube. If bile or pigment be present, the gravitated chloroform will be colored yellow.

*Gmelin's test* is most commonly employed, though it is not the most delicate. A few drops of urine and nitric acid are allowed to run together on a white porcelain plate; if bile-pigment (bilirubin) be contained in the urine, a play of colors ensues, the green predominating, followed by the blue, violet, and red, each shade representing a new form of pigment. The first color noticed (green) corresponds to the biliverdin or normal bile-pigment of herbaceous animals. This oxidation of bilirubin into biliverdin is better accomplished by nitric acid containing a little nitrous acid. Hence, the test may be improved by adding enough fuming nitric to ordinary nitric acid to form a yellow trace of the nitrous acid. This may be placed in a test-tube or wine-glass, and some of the urine added gently from a pipet. Bile-pigment will be indicated by successive rings of green, blue, violet, and red from above downward; this occurs, however, only when the bile-pigment is present in considerable quantities.

*Rosenbach's test* is a modification of Gmelin's, and is more distinct. The urine is first filtered, and a drop or two of the nitric-nitrous acid is then poured upon the filter-paper, when the characteristic colored rings will appear if bile be present. According to Penzoldt, the Gmelin-Rosenbach test is made more distinct by acidulating the filtrate with



acetic acid and pouring a thin layer into a white shallow dish. The acetic acid assumes a greenish-yellow, and later a green, or even a blue-green, shade if bile be in the urine. This reaction is quickened or intensified by the application of heat to the liquids.

In the *Marechal-Rosin test* a mixture of one part of the tincture of iodine and ten parts of alcohol is spread in a deep layer over the suspected urine in a test-tube or glass. A grass-green ring forms at the point of contact in choluria.

**Bile-acids.**—These are principally the glycocholic and taurocholic acids. Traces are found in normal urine, and their clinical significance or diagnostic importance, as far as is known, is practically *nil*.

When testing for bile-acids the Stranburger modification of Pettenkofer's method may be used, as follows: "After isolation cane-sugar is added to the extract, which is then filtered. A drop or two of strong sulphuric acid is spread on the dried filter; a violet or purple color appears" (Musser).

Other constituents of the urine in choluria of long standing are slight quantities of albumin and icteric or yellow bile-stained hyaline or finely-granular casts.

A point in **differential diagnosis** should be noted in connection with the fact that *certain drugs*, as rhubarb and santonin, when given internally, may produce a discoloration of the urine similar to that caused by the presence of bile. On agitation, however, there will be no yellow foam and no reaction to the tests for bile, while the addition of liquor potassæ causes a red color.

#### UROBILINURIA.

**Definition.**—The presence of pathologic quantities of urobilin in the urine. Urobilin is the principal coloring-matter of the urine, and hence is present in normal urine in small quantity. It is derived from hematoidin or bilirubin as a product of the reduction of these substances in the tissues and blood-vessels.

When present in large quantities urobilin gives to the urine a red-brown color. This is seen in fevers, varying in depth of shade according to the degree of pyrexia; also in diseases of the liver, after hemorrhagic effusions (due to resorption), in the hemorrhagic diathesis, in purpura, and in progressive pernicious anemia.

When deposited in the tissues it gives rise to a form of jaundice—in which there is a brownish discoloration of the skin—called *urobilin-icterus*.

**Diagnosis.**—The presence of urobilin is best detected by a spectroscopic examination. A marked absorption-band between Fraunhofer's lines (f and b), fading off from the green into the blue, is characteristic. Chemically, the addition of a few drops of a watery solution of zinc chlorid to the urine will cause the peculiar red-green fluorescence of urobilin to appear.

#### GLYCOSURIA.

**Definition.**—The presence of sugar (glucose) in the urine. Normally, a trace of sugar is present in the blood (glykemia), but it may be doubted whether any is excreted in the urine in health, except after

the ingestion of an excess of food rich in saccharine or starchy substances. Uric acid may give the same reactions as glucose in the urine.

**Etiology.**—The causes of glycosuria may be enumerated as follows: (1) Diabetes mellitus—the most common. (2) Certain diseases, like gout (*intermittent glycosuria*), cholera, typhoid, typhus, and scarlet fevers, whooping-cough, diphtheria, malaria (*paroxysmal glycosuria*), tetanus, phthisis, hepatic cirrhosis, and organic nervous diseases, especially those affecting the medulla and involving the floor of the fourth ventricle. Glycosuria may also result from psychic causes, as excessive mental exertion, extreme emotional activity (grief, worry, and shock), from injuries, as cerebral concussion and hemorrhage, and fracture of the skull, from apoplexy, cerebro-spinal meningitis, and after epileptic paroxysms. (3) Pregnancy. (4) Certain toxic agents cause a transient glycosuria, among these being carbon monoxid, morphin, hydrocyanic acid, amyl nitrite, curare, chloral, alcohol, mercury, arsenic, turpentine, phloridzin, and various coal-tar derivatives, as salicylic acid and salol. This source of glycosuria has been experimentally demonstrated in dogs by Paul Gibier of the New York Pasteur Institute. (5) Obesity and thyroidismus may cause a temporary glycosuria (lipogenic). (6) Pancreatic disease (chronic interstitial pancreatitis and, less commonly, pancreatic calculi, carcinoma, and cysts). (7) Glycosuria may occur in exophthalmic goiter, and, according to Lyman, may be present for a short time in (8) diabetes insipidus. (9) Heredity probably plays a part in predisposing to glycosuria in certain cases, particularly in the permanent affection. (10) Dietetic or alimentary glycosuria may at times be noted, as in beer-drinkers.

**Diagnosis.**—The daily quantity of the urine of typical glycosuria—*i. e.* when masking saccharine diabetes—is greatly increased (60 fluid-ounces—2 liters—and over *per diem*); it is of high specific gravity (1025 and over), of a clear, pale-yellow color, a “ripe-fruit” odor, a sweetish taste, and an acid reaction that is intensified on standing, owing to the fermentation of the sugar. Albuminuria is not infrequently associated with glycosuria, and the albumin should be removed before testing decisively for sugar. Again, since urine of high color, heavy density, and marked acidity often contains uric acid, and since this substance, as already pointed out, responds to the sugar tests, care must be exercised lest a false conclusion be drawn.

**Tests.**—The most important of these depend mainly upon the peculiar property of glucose in reducing the blue oxid of copper to the orange or red suboxid. It must be remembered that other metallic substances are similarly decomposed.

(1) *Fehling's Test.*—Two solutions are used, equal parts being mixed to form the Fehling's solution, as follows:

Solution I. contains 34.64 gm. of cupric sulphate, dissolved in enough water to make 500 c.cm. Solution II.: 173 gm. of Rochelle salt are dissolved in 480 c.cm. of sodium hydroxid (sp. gr. 1.14); this is then diluted with water up to 500 c.c.

Application: Dilute 1 c.c. of Fehling's solution (about 10 drops of each of the above solutions) with about 1 dram (4 c.c.) of water in a test-tube, and heat to the boiling-point. If the clear blue color remains, the solution is ready for use; should it change color, however, the solution is unfit for use and should be discarded. The suspected

urine is added, drop by drop, heating occasionally, when, if glucose be present, the blue color will be discharged by a yellow turbidity, which increases until finally a deep-yellow or orange red precipitate falls. Bluish-white flakes and a greenish discoloration of the mixture simply indicate cupric hydroxid, and not glucose. This test serves for the detection of .001 per cent. of glucose (Wormley).

(2) *Trommer's Test*.—To about 5 c.c. of urine in the tube add one-third or one-half its volume of potassium or sodium hydroxid, and then, drop by drop, add a 10 per cent. solution of cupric sulphate. If a bluish-white precipitate falls, either filter or agitate the liquid until it assumes a slight and uniform turbidity; then heat, and, if sugar be present, a yellow or red deposit of cuprous oxid falls: .01 per cent. of glucose may be detected in this way.

Besides uric acid, there are certain other substances which when present in urine make the copper tests fallacious by reducing the cupric to cuprous oxid. Among these are mucin, lactose, pyrocatechin, hydrochinon, bile-pigments, glycosuric acid, the products of elimination after the ingestion of chloral (urochloric acid), and benzoic and salicylic acids.

(3) *Böttger's Bismuth Test*.—This may be performed as a counter to the copper tests. Albumin, however, interferes with the test on account of the contained sulphur, which forms a black bismuth sulphid: hence, if present, it must first be removed. This may be done by acidulating the urine with acetic or nitric acid, boiling, and then filtering. Böttger's test is then made by adding to the non-albuminous urine or to the filtrate from one-half to an equal quantity of liquor potassæ and a few grains of bismuth subnitrate. Boil for several minutes, and if glucose be present black metallic bismuth will be precipitated.

(4) *Nylander's reagent* may be employed. This consists of 2 parts of basic bismuth nitrate and 4 parts of sodium tartrate to 100 parts of an 8 per cent. solution of caustic soda. One part of the reagent is boiled with 10 parts of the urine for a few minutes, when a change from the original to a brown or black color will indicate the presence of glucose. This test is quite distinct, but has the fallacy that is common to all the bismuth tests, of forming a black precipitate with the sulphur compounds.

(5) *Fermentation Test*.—Though not always convenient to apply, this is, nevertheless, a most reliable test. It depends upon the action of yeast in breaking up glucose into alcohol and carbonic-acid gas (carbon dioxide). It is performed easily by adding a small piece of compressed yeast to the urine in a test-tube, inverting the latter in a dish of the same, and standing aside for twelve to twenty-four hours, the temperature being kept at about 80° to 100° F. (26.6°–37.7° C.). The evolution of gas resulting from the fermentation of the sugar takes place, with a consequent reduction of the specific gravity of the urine. The yeast may be tested simultaneously for its purity and strength by placing one portion in a test-tube containing about two-thirds mercury and filling with normal urine, and a similar portion in a second tube with mercury and a thin, watery solution of sugar or glucose; the fermentation test of the suspected urine may be made at the same time, and all three tubes inverted over a dish of mercury. Obviously, the first



tube should not show the presence of carbon dioxid if the yeast was free from sugar ; but the second tube should show this gas to be present or the yeast was inert.

Other tests, such as *Moore's liquor-potassæ-and-boiling test*, *Johnson's picric-acid test*, and the *phenyl-hydrazin test*, are more intricate and in no way more reliable.

The *quantitative estimation of sugar* may be made with Fehling's solution in two parts, as recommended above for the qualitative test. This method is based upon the fact that the cupric oxid of 1 c.c. of Fehling's solution will be reduced by not less than 0.005 gm. of glucose. Place 1 c.c. of the solution in a test-tube and dilute with 4 c.c. of water (5 c.c. dil. sol.). Heat to the boiling-point, and add 1 c.c. of urine, and heat the liquid again. If reduction has taken place, 0.005 gm.—0.5 per cent. or more—glucose is present ; if no reduction has occurred, less than 0.5 per cent. is present. If 2 c.c. urine are used before the color of the Fehling solution is discharged, there will be 0.25 per cent. glucose. If  $\frac{1}{2}$  c.c. is used, 1 per cent. is present. If  $\frac{1}{10}$  c.c. urine is all that is required (about 2 drops), then 5.0 per cent. of glucose is present.

*Roberts' differential-density method* depends upon a loss in the specific gravity of the urine, due to the fermentation of glucose. According to Roberts, each degree in specific gravity lost is equivalent to 1 grain of glucose in 1 imperial fluidounce (437.5 gr.) of urine, or one degree represents 0.23 per cent. glucose. (See works on Urinalysis.)

*Circumpolarization*.—Finally, sugar may be determined by the saccharimeter or polariscope. Glucose polarizes light to the right. The percentage may be calculated by reading the vernier scale indicating the degree of reflection, and multiplying the number read by the factor of the apparatus used, after making any required corrections.

#### ACETONURIA, DIACETONURIA, AND OXYBUTYRIA.

Acetonuria, diacetonuria, and oxybutyria are so closely allied with glycosuria, and especially with diabetic coma (acetonemia), that they may be considered together. In the first-named condition the urine contains acetone ; in the second, diacetic or aceto-acetic acid ; and in the last, oxybutyric acid. Diacetic and oxybutyric acids are products of the decomposition of acetone, and hence the importance previously ascribed to the latter, when detected in the urine of diabetics, has given place to the two former, the oxidation of which yield acetone.

**Acetonuria** may exist to a minute degree in health, the acetone being a product of the normal metamorphosis of albumin. It may be present also in—(1) diabetes ; (2) carcinoma ; (3) febrile conditions ; (4) inanition ; (5) psychoses ; and (6) auto-intoxication. Urine that contains acetone in pathologic quantities has a fruity (apple-like) odor or one resembling that of chloroform.

**Tests**.—(1) *Gerhardt's* original test consisted in the addition of a few drops of the tincture of the chlorid of iron, which produced a Burgundy-red color with acetone, or rather with the aceto-acetic acid.

(2) *Nitro-prussid Test*.—To a fluidounce (32.0) of the urine add a dram or two (4.0–8.0) of a solution of sodium nitro-prussid (gr. v. to 3j

—0.324 to 32.0) and a few drops of strong aqua ammoniæ. On standing a rose-violet color appears. According to Legal, proportionately smaller quantities of urine and the reagent may be used, and strong liquor potassæ. A bright-red color develops, and fades rapidly, but upon adding acetic acid this changes to purple or violet-red (Vierordt). This is a better test.

(3) Perhaps the most accurate and, at the same time, satisfactory test for acetone is the following: Distil the urine with a little phosphoric acid, and add to the distillate a few drops of sodium hydroxid and of Lugol's solution. If acetone be present, yellow crystals of iodoform will form, with the characteristic odor.

**Diacetonuria** and **oxybutyria** never occur normally. They are often associated with acetonuria in diabetes, and sometimes in fever, or occur as an independent disease (V. Jaksch). Moreover, it is believed that diacetic and oxybutyric acids are the causes of diabetic coma, and not acetone, as was held formerly. Stadelmann affirms that of like value with the recognition of oxybutyric acid in diabetes is the determination of a marked and increasing amount of ammonia in the urine (1 gram—gr. xv—and more *per diem*), as indicating the imminence of diabetic coma. Diacetonuria is found to occur in certain acute diseases of children, accompanied with convulsions.

**Tests.**—The presence of diacetic acid is demonstrated by the chlorid-of-iron reaction, as in the case of acetone, except that the urine is boiled previously. This is done to avoid fallacy, since in unboiled urine acetic, formic, and oxybutyric acids may strike a Burgundy-red also; in urine that has been previously boiled these do not react, while the diacetic acid does, if present. Diacetic acid is usually present simultaneously with acetone. If a portion of the urine is mixed with sulphuric acid and extracted with ether, diacetic acid may be inferred to be present if the extract shows a chlorid-of-iron reaction that fades within twenty-four hours (V. Jaksch).

#### LITHURIA.

**Definition.**—A persistent excess of uric (lithic) acid and urates (lithates) in the urine.

Normal urine contains about 0.4 part of uric acid to 1000 parts of urine (about gr. x—0.648—*per diem*), or it exists in the proportion of about 1 to 45 of urea, the principal solid constituent. The evidence of recent experimentation is against the view that uric-acid formation is to be regarded as an "unfinished product" in the old sense.

**Etiology.**—The causes of lithuria, as seen in certain conditions in which this metabolic change occurs, may be put down to be chiefly as follows: (1) Lithemia (uricemia; uric- or lithic-acid or gouty diathesis); (2) gout and rheumatism; (3) fever; (4) leukemia and pernicious anemia; (5) pulmonary affections in which the interchange of gases is interfered with; (6) a highly nitrogenous diet. Certain other conditions of the urine may diminish its power of dissolving the uric acid shortly after voidance, and may cause a deposit that should not be mistaken for an excess. Such are—(a) temporary increase in the quantity of uric acid from an over-indulgence in nitrogenous food; (b) temporary high acidity; (c) deficiency in mineral salts.

**Diagnosis.**—The urine has a high specific gravity, a deep red-yellow color, and a marked acid reaction, although, rarely, uric acid is formed in neutral or alkaline urine (Vierordt). Albumin may be present in small amount at the same time. On standing the uric acid is deposited in yellowish-red or “Cayenne pepper” grains, composed of microscopic uric-acid crystals. Chemically pure uric acid is colorless, but that deposited from urine has always this yellowish-red appearance both to the naked eye and under the microscope. Examination with the latter shows a great variety of rhombic prisms—“whet-stone-shaped,” “crosses,” “lozenges,” and other many-shaped and sized crystals—single and in agglomerations.

**Test.**—The *murexid reaction* may be obtained by evaporating a little urine in a watch-glass or porcelain dish, adding a few drops of strong nitric acid, and heating to dryness again; this is allowed to cool, and a drop of liquor ammoniæ added, when a beautiful purple shade of murexid will appear if uric acid be present.

**Urates.**—These are increased in pathologic conditions that give rise to uric acid in excess, and are usually present with the latter in some quantity. It is not rare, however, in healthy individuals for a deposit of urates to occur in concentrated urine exposed to a cool atmosphere. Urates appear also in the scanty urine due to the profuse perspiration and diarrhea of renal congestion, in fever, from renal calculi, and after a meal rich in albuminous elements.

Urates occur principally as acid sodium urate, calcium urate, and ammonium urate. They appear macroscopically as a flesh-colored or “brick-dust” (lateritious) sediment; this is usually abundant and very finely granular in appearance, while the urine above is cloudy. It is quite characteristic that upon heating such urine it becomes clear, the urates being completely dissolved. Microscopically, the sodium and calcium salts of uric acid occur as needle- or dumb-bell-like crystals or as fine, dark, amorphous granules. Ammonium urate is found in alkaline urine, often with triple phosphates when some putrescence has ensued. It is seen in dark-brown or green spiculated spherules; these are sometimes called “hedge-hog” or “thorn-apple” crystals. On the addition of a drop of hydrochloric acid under the cover-glass uric-acid crystals may be seen to develop.

#### OXALURIA.

**Definition.**—A persistent excess of calcium oxalate in the urine. A few crystals may occur in normal urine that has been standing for a long time.

*Transient oxaluria* may follow the ingestion of sub-acid fruits, as pears, or of vegetables containing oxalates, as rhubarb, tomatoes, sorrel, and cauliflower.

**Pathology.**—Oxaluria has been described by some English physicians as an independent disease or special diathesis in which marked dyspepsia and hypochondriasis or neurasthenia are associated. The condition is better explained, probably, as one of a disturbed metabolism—particularly of the fats and carbohydrates—in which the oxaluria and the nervous symptoms are manifestations analogous to the lithuria and the



irregular gouty symptoms of lithemia. Oxalates and lithates are not infrequently found together in the urine of those subject to the gouty habit. Oxaluria is also present in wasting diseases, as in tuberculosis and diabetes mellitus, and in the cancerous cachexia; it may appear in catarrhal jaundice, spermatorrhea, also with the "mulberry calculi," and in general paresis of the insane. Slight albuminuria is not infrequently associated.

**Diagnosis.**—Oxalate-of-lime crystals appear in the urine in two forms—most commonly as minute, regular, highly-refracting octahedra, or, more rarely, as hour-glass- and dumb-bell-shaped crystals.

The octahedral crystals have two crossed axes, giving a star or envelope-like appearance. Oxalates sometimes give a glittering and scintillating effect to floating mucus in urine that has undergone fermentation.

The **prognosis** is usually favorable.

In the **treatment** of oxaluria the nitro-hydrochloric acid in 2-drop doses is a useful agent.

#### PHOSPHATURIA.

**Definition.**—A persistent excess of phosphates in the urine.

Phosphoric-acid salts may be precipitated in normal urine that has become temporarily alkaline. These acid sodium and potassium phosphates in normal acid urine are derived from the alkaline phosphates (neutral sodium and potassium phosphates) of the blood. In normal urine 1.2 parts of alkaline phosphates per 1000 and 0.8 part of earthy phosphates are appreciable.

**Etiology and Pathology.**—Conditions that produce an alkaline fermentation of the urine cause a deposit either of *amorphous earthy phosphates* or of *crystalline phosphates*. They are also found in the decomposing urine of chronic cystitis, of phosphatic vesical calculi, of paralysis, and in undue retention of urine. In this alkalinity, due to the ammoniacal fermentation of urea, ammonium carbonate reacts with the phosphates of magnesium to form the triple ammonio-magnesia phosphatic crystals, the commonest variety of phosphaturia. Here the phosphates are deposited before or immediately after the urine is passed, giving a milky appearance to the last portion. Deposits of phosphates, and especially of triple phosphates, by no means, however, indicate an actual phosphaturia. This must be determined by chemical analysis. *Amorphous carbonate of lime* in small quantity may be present also if the urine is strongly alkaline and ammoniacal (Beale). The *calcium phosphates* are generally more abundant than the magnesian, and may be found in cases of nervous or atonic dyspepsia, neurasthenia, melancholia, and other debilitated conditions. Whether or not a marked precipitate of phosphates means any detriment to nervous tissue alone has not been determined precisely as yet. Obviously, however, a certain portion of phosphates is supplied by the food, and the rest by the body owing to defective assimilation and metabolism.

A quantitative estimation of the daily output of phosphates shows a decided increase in wasting diseases, as tuberculosis, leukemia, chronic articular rheumatism, and acute yellow atrophy of the liver. The phosphoric acid is not increased, however. The so-called "phosphatic diabetes" is characterized by polyuria, excessive phosphaturia, thirst, emaciation, and nervous disturbances (Tessier).

**Diagnosis.**—Phosphatic urine has usually a stale, ammoniacal odor, a whitish turbidity, and a copious light-colored granular sediment falls on standing. Microscopically, the calcium phosphate crystals appear singly as “knife-blade,” “arrow-head,” or “slender wedge-shape,” or in stellate clusters. Acetic acid dissolves them. The ammonio-magnesian phosphate crystals are transparent rhombic or triangular prisms, large and small—“coffin-lid-shaped.” These also are soluble in acetic acid; oxalate-of-lime crystals are not so.

On heating phosphatic urine an increased cloudiness is produced that simulates albumin, but on acidifying, as with a drop of nitric acid, this is cleared up at once.

#### LEUCINURIA AND TYROSINURIA.

**Definition.**—The presence of leucin and tyrosin in the urine. These are strictly pathologic substances, and are usually found together. They are products of the decomposition of albumin, intermediary to the formation of urea, and are most apt to be found in the urine, along with biliary matters, in certain hepatic conditions.

**Etiology.**—The principal causes of leucinuria and tyrosinuria are acute yellow atrophy of the liver, acute phosphorus-poisoning (in both of which fatty degeneration of the liver is pathologically conspicuous), specific infectious diseases, as typhoid fever, small-pox, and yellow fever, and pernicious anemia. Leucin and tyrosin have also been found (Kirkbride) in the urine of a patient with erysipelas, during two days of the decline.

**Diagnosis.**—Of the two substances, leucin is the more soluble, and hence is rarely found in the urinary sediment. Tyrosin, on the other hand, may be discovered sometimes as a fine greenish-yellow deposit. Bile-pigment may be found not infrequently in urine containing leucin and tyrosin. A trace of albumin also may be present, while the urea is, as a rule, markedly diminished. Leucin and tyrosin may be detected by evaporating a few drops of urine on a glass slide and examining microscopically. Leucin appears in the form of slightly glistening, greenish-yellow spheres that may show radiating lines and concentric rings. Tyrosin is recognized by the slender tufts of fine, needle-like crystals arranged in star- or cross-like fashion.

If the residuum after evaporation be heated with a drop of nitric acid, slowly evaporated to dryness, and then touched with a drop of sodium hydroxid, the leucin, if present, will assume a yellowish-brown hue.

Tyrosin becomes red in color when boiled with Millon's reagent of mercurous nitrate, or it is demonstrated by a violet color when carefully warmed with a little sulphuric acid, and then treated with a drop of the solution of phenic chlorid.

#### CYSTINURIA.

**Definition.**—The presence of an excess of cystin in the urine. Cystin in minute quantity may be found sometimes in normal urine. It contains sulphur. The *causes* of cystinuria have not been well made out, though hereditary influences seem to have an important bearing on the etiology; in what manner they act, however, is not known. On account of the insolubility of cystin any marked quantity would be deposited in

the urine. Cystin calculi sometimes result, though cystinuria may exist without the presence of a cystin calculus.

Brieger points out a probable significance in the discovery of the associated presence of ptomains with cystinuria. Thus, in certain infectious diseases, as intestinal mycosis, a ptomain-cystinic product is supposed to be formed, then to be absorbed, and finally decomposed in the urine, thus setting free the cystin. Cystitis may be caused by the action of the ptomains.

**Diagnosis.**—The sediment is light, and not very unlike that of the amorphous urates. It is not dissolved by heat, however, though soluble in ammonia. Under the microscope cystin occurs in the form of thin, transparent, hexagonal crystals. Care should be exercised in forming a diagnosis of cystinuria that a contamination with iodoform be excluded, since the microscopic appearance of that substance is similar to that of cystin. On account of the sulphur contained in cystin, a test may be employed by which hydrogen sulphid is liberated, as by boiling the suspected urine with a solution of lead oxid and sodium hydroxid, black lead sulphid resulting from the reaction if cystin be present.

#### VARIOUS OTHER CONDITIONS.

**Urea.**—This occurs in solution in the normal urine as a product of the perfect decomposition of the nitrogenous elements of food and tissues. In 1000 parts of urine about 20 parts are constituted of urea (2 per cent., equivalent to about gr. 450—30.0—daily). The quantity of urea is *increased* in the urine after the ingestion of a considerable quantity of proteid food; sometimes after exertion; in acute inflammation and in fevers—either relatively or absolutely, as in pneumonitis; in diabetes and other morbid conditions in which metabolism is accompanied by an increase in the tissue-waste. In febrile states its excretion increases or diminishes with the exacerbations and remissions of temperature respectively.

Urea is pathologically *diminished* in quantity in all forms of nephritis, and markedly so in uremia; in organic liver-diseases, as acute yellow atrophy; in cachectic and anemic states; and in dropsy, inanition, and allied conditions.

The quantitative estimation of urea may be made according to one or more of several methods: Fowler's hypochlorite test (with Labarraque's solution) is perhaps the most practical for ordinary clinical purposes; the hypobromite and Liebig's methods, both requiring special apparatus, are better adapted for the laboratory.<sup>1</sup> Fowler's method is based upon the loss of specific gravity upon the liberation of the nitrogen of the urea. The mean specific gravity of a mixture of 1 part of urine and 7 parts of the solution of sodium hypochlorite is taken while quiescent, and is then subtracted from the specific gravity of the mixture taken after agitation several times during about two hours. The difference which is due to the liberation of the nitrogen (as is shown by the effervescence), multiplied by the factor 0.77, gives the approximate percentage of urea in the urine. This test, however, has a considerable range of error.

<sup>1</sup> See works on Urinalysis.



Urine evaporated to a syrupy consistence and then treated with nitric acid shows crystalline quadratic plates of urea nitrate.

**Chlorids.**—About 10 parts of the chlorids of sodium and potassium in 1000 parts of urine are excreted daily. They are *increased* in the urine after muscular exertion, during the resorption of mechanical or inflammatory transudations and exudations, and in intermittent fevers, owing to the destruction of the red corpuscles.

Pathologic *diminution* in the quantity of chlorids occurs in fevers, in the nephritides, in cachectic conditions, and especially in such diseases as pneumonitis, pleuritis, and rheumatism. In the last-named class the chlorids diminish as exudation continues, and may even totally disappear from the urine in extensive pneumonic consolidations, to reappear again with the resorption of the exudate.

**Test.**—The chlorids may be detected, after first removing any albumin that may be present, by acidulating with a few drops of nitric acid (to keep the phosphates in solution), and by then adding, drop by drop, a strong solution of argentic nitrate. According to the abundance of the resultant white, curdy precipitate of argentic chlorid a rough estimate may be made of the total quantity of chlorids in the urine.

**Lipuria** is a term applied to the presence of fat in the urine. It may result from the steady use of cod-liver oil or of fatty food, or it may be found in pyonephrosis (Ebstein); in phosphorus-poisoning; in prolonged suppuration; in the lipemia of diabetes mellitus; in the “large white kidney” with fatty degeneration of chronic Bright’s disease; in beer-drinkers; and in chyluria. Fatty urine becomes clear upon agitating after the addition of ether.

**Lipaciduria**, or urine containing volatile fatty acids (acetic, butyric, and propionic), is as yet without diagnostic significance.

**Melanuria**, or urine containing the pigment melanin, is found in cases of melanotic sarcoma. The urine is dark, either just after being voided or after some exposure and oxidation.

**Hematoporphyrinuria** (*Urospectrin*).—This term implies the presence of hematoporphyrin (iron-free hematin) in the urine. It occurs after long-continued use (even in small doses—Müller) of certain coal-tar products, particularly sulfonal and trional. In addition to the gastric and nervous *symptoms* in poisoning from these substances is a cherry-colored or dark blue-red urine, the abnormal appearance of the latter being due to the presence of hematoporphyrin resulting from the destruction of the red blood-corpuscles. The condition has proved fatal in several cases where albuminuria showed the kidneys to have been diseased. The urine is always quite acid. According to Garrod, hematoporphyrin is a scanty though constant ingredient of normal urine. He extracts it by adding 100 c.cm. of urine to 20 c.cm. of a 10 per cent. solution of sodium hydroxid. This precipitates the phosphates, which are washed with water and redissolved with rectified spirits. After acidulation with hydrochloric acid the solution shows spectroscopically bands of acid hematoporphyrin. The *treatment* consists in the prompt withdrawal of these drugs and the free administration of alkalies.

**Pneumatinuria**, or gas-formation in the bladder, rarely occurs. Heyse<sup>1</sup> records a case of myelitis in which this condition was present.

<sup>1</sup> *Zeit. f. klin. Med.*, 1894, xxiv. p. 130, quoted in *The American Year-Book of Medicine and Surgery* for 1896.

**Fibrinuria.**—In certain conditions of the genito-urinary tract, particularly pyelitis and ureteritis, fibrinous (and mucous) casts are found in the urine. Fibrinuria may follow nephro-lithiasis, as in a case recorded by v. Jaksch.

**Bacterinuria.**—There are probably few specimens of urine that do not contain bacteria. Engel has found a great variety of organisms in the nephritides, one of which (a micrococcus of characteristic growth and properties) was present in 17 out of 31 cases, hence regarded by him as the specific cause of some of the cases in this category of diseases. This organism was found in mild types of nephritis, and Engel believes it to be responsible for many instances of the sort beginning as mild forms of "*bacterial albuminuria*."

**Lactosuria.**—Lactose is found in the urine of some puerperæ.

**Inosituria.**—Inosite occurs in the urine in diabetes insipidus.

**Alkaptonuria.**—Alkaptone is an obscure substance (so called by Bredeker) that is sometimes found in the urine of phthisical cases, or at times in that of patients without any apparent local or general disease. Alkaptonuria seems to be congenital in a few cases. On exposure the urine darkens in color; also upon the addition of liquor potassæ. It gives the sugar-reaction with Fehling's solution (Osler).

Urine as affected by the administration of various drugs—as carbolic acid, salol, antipyrin, and potassium iodid—responds to certain chemical tests, for the study of which the reader is referred to works on urinalysis and clinical diagnosis.

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## THE NEPHRITIDES.

BEFORE considering the several varieties of nephritis, and especially the clinical history peculiar to each variety, it may be well to first describe certain general manifestations of renal diseases that are more or less common to all. Reference to these symptoms under the different forms of nephritis will, it is hoped, thus make possible a clearer apprehension of their significance and clinical importance, as well as render unnecessary any further elaboration.

One of these pathologic conditions has already been described—viz.

- (1) *Albuminuria*. It remains, then, to speak of (2) the *Morphologic constituents* of the urine in nephritis, (3) *Edema (anasarca, dropsy)*, and (4) *Uremia*.

### THE MORPHOLOGIC CONSTITUENTS OF THE URINE IN RENAL DISEASE: CASTS, EPITHELIUM, ETC.

1. **Tube-casts.**—These are undoubtedly the most important morphologic elements in the urine of a nephritic. Albuminuria is coincidentally present, and the occurrence together of these two pathologic constituents furnishes indisputable evidence of renal disease. According to the nature and quantity of the casts also may be determined the character and variety of the affection of the kidneys in most instances. Casts, as their name implies, are simply cylindric bodies moulded in

the renal tubules, and composed essentially of the coagulable substances in the serum exuded from the blood-vessels. The coagula of the tubules are mostly albuminous. Other morphologic elements may be mixed with casts, such as epithelium, red blood-cells, pus-cells, and the granular matter and fat-droplets due to degeneration of the renal epithelium.

Singly, the casts are invisible to the naked eye, but in acute nephritis they may be so abundant as to form a cloudy sediment near the bottom of the urine-containing glass.

(a) Microscopically, the *unmixed* or *hyaline* cast—the commonest—appears either long or short and narrow or broad, of a clear, transparent, homogeneous substance, delicate in outline, and often showing ends with a cheesy—or wax-like—fracture. They may be straight or slightly curved and tortuous, with fine short transverse lines here and there at the borders of the cast. Rarely, a cast may be found equal to a millimeter in length. The so-called *narrow casts* are about equal in width to the diameter of a leukocyte, while the *medium* or *broad casts* are from three to four times this size. They will take either the carmin or gentian-violet stain. Hyaline casts are usually associated with other varieties of casts in nephritis, though in fevers, congestion of the kidneys, chronic interstitial nephritis, and in amyloid kidney they may occur unassociated with other forms of casts (Vierordt).

(b) *Granular* casts are nothing more than hyaline casts with fine or coarse granules superadded. The granules represent minute, opaque particles of urates, albumin, fat, cellular debris, and even bacteria (*bacterial casts*). It should be remembered, however, that granular casts may be simulated by casts of coagulated albumin covered with particles of hematoidin, especially in acute nephritis. The hematoidin can be recognized, however, by the brown-yellow coloration.

(c) *Epithelial* casts are hyaline casts more or less covered with renal epithelium, indicating an acute desquamative nephritis. The epithelial cells may show evidence of granular or fatty change.

(d) *Blood-casts* consist of soft hyaline casts having red blood-cells imbedded in them. These are present in renal hemorrhage and in acute hemorrhagic nephritis.

(e) *Waxy* casts are similar in appearance to hyaline casts, though better defined, broader as a rule, and of an opaque, slightly yellowish tint. They often show broken ends. They do not necessarily indicate amyloid disease of the kidney, as was formerly held. They may, however, sometimes show the amyloid reaction with iodine and potassium iodide, and are always suggestive of serious renal disease.

(f) *Fatty* casts are such as have left upon and in them fat-droplets or granules, which, if abundant, are indicative of fatty degeneration of the kidney. Cells showing granulation from fatty change may be seen simultaneously.

Rolled casts or *pseudo-casts* (made by sliding a cover-glass over a specimen of urine) of urates, epithelial cells, blood-corpuscles, and debris should not be mistaken by beginners for genuine tube-casts. The same may be said of cylindroids, mucous cylinders, and foreign substances. Nephritis may exist when the casts are always to be found, varying in numbers only, while albuminuria may be inconstant or intermittent.

2. **Epithelium.**—Renal cells are found in the urine of those forms of



nephritis that are characterized by a catarrhal or desquamative and exudative process in the tubules. Epithelial cells from the kidney are polygonal or spheric in contour, with an indistinct cell-wall; they have a large oval nucleus, and are either abundantly granular or show a fatty change. These cells are about the size of the white corpuscle.

3. **Leukocytes.**—Only when attached to casts can it be positively affirmed that leukocytes are of renal origin (Strümpell). The pus-cells are frequently seen to be without nuclei in marked or chronic pyuria.

4. **Red Blood-corpuscles** (*vide* Hematuria, p. 934).—In acute hemorrhagic nephritis and in severe renal congestion free red blood-corpuscles are generally to be found.

5. **Fat-globules and granular, fatty-degenerated cells** are seen especially in the subacute and chronic forms of nephritis with fatty degeneration of the proliferated epithelium, or in the fatty stage of large white kidney.

#### DROPSY OF RENAL DISEASE.

Since, as in other conditions, renal dropsy or edema is an abnormal accumulation of watery fluid transuded from the blood-vessels into the cellular tissues and lymph-spaces, the question arises, "What is the rationale of its development in nephritis?" On the ground that the renal secretion consists principally of water, and that in most forms of nephritis the urine is diminished, it was formerly held that the dropsy was due to the saturation of the tissues with the water that was not excreted by the kidneys. This theory is not fully tenable, however, for there are some cases of edema unaccompanied by any diminution in the daily quantity of urine; on the other hand, certain instances of renal disease in which there is a state of almost anuria show no evidence of dropsy whatever. Indeed, it has been suggested (Strümpell) that edema is the cause rather than the result of a diminished elimination of water by the kidneys, this view corresponding in part with Cohnheim's assertion that the increased transudation is due to changes in or injury to the endothelium, increasing the perviousness of the blood-vessels. The failure of any one theory as advanced above to explain the etiology and pathology of edema has justified the proposal of another and undoubtedly a more plausible one by Landerer—*viz.*, that the relaxation of the tissues (which may be caused by the increased transudation of stasis, or by hyponutrition from hydremia), and their consequent loss of elasticity, prevent that forcing of the lymph into circulation that exists in the normal state, and as a result a watery infiltration of the tissues is permitted. The loss of elasticity or power of resistance in edematous tissues is quite apparent under the skin, and affords a positive means of diagnosis in the pitting produced by the pressing finger.

The dropsy of the nephritides may be either slight or marked, local or general (anasarca), and sudden or slow in onset. It is purely renal in origin perhaps only in acute Bright's disease or in the earlier stages of chronic Bright's disease. In all forms of chronic nephritis the dropsy may be due, in part, to the venous stasis of cardiac incompetency. In chronic interstitial nephritis, especially, edema is slight, and usually is the result of weakness and dilatation of the heart, increasing *pari passu* with the latter.

I desire to mention here those rare cases of dropsy that simulate Bright's disease in which no satisfactory causative lesion is apparent or discoverable, and also those cases, rarer still perhaps, that have a peculiar family or congenital origin.

**Physical Signs.**—The recognition of edema is made possible by both *inspection* and *palpation*. Renal dropsy is manifested first by puffiness of the skin of the face, and especially of the eyelids. At other places where there is loose subcutaneous cellular tissue, and in particular where the parts are dependent, dropsy is most apt to be seen early, as under the malleoli of the ankles, the dorsum of the foot, and the scrotum. Later, the limbs and the lower part of the back become swollen, and even the whole body is involved in severe cases. The skin has a peculiar waxy pallor and a glossy appearance. When evident vascular or cardiac changes exist, so as to permit of increased dropsy from engorgement, as in cirrhotic kidney, a cyanotic or muddy color of the skin may prevail.

**Pathologic Features.**—Dropsy is most constant and most persistently decided in the large white kidney of subacute or chronic nephritis: it is most uncommon and irregular in chronic interstitial nephritis (contracted kidney). The familiar pitting on pressure over edematous tissues is a true indication of fluid under the skin. There is also a doughy or putty-like consistence. In very marked cases of dropsy the deeper parts, such as the muscles, become affected. The serous cavities also in general anasarca show evidences of effusion, and thus give rise to hydro-thorax, hydro-peritoncum, and hydro-pericardium. Less frequently there may be edema of the larynx, uvula, conjunctiva, and other mucous membranes. Edema of the brain, either local or general, may be the cause of grave uremic symptoms in chronic nephritis, or of unilateral convulsions or paralysis and apoplectic seizures. The dropsical liquid is chemically similar to a diluted blood-serum. A minute quantity of albumin and urea is present.

#### UREMIA.

**Definition.**—Uremia is the term applied to a group of manifestations, mainly nervous and either acute or chronic, resulting from a toxemia due to the retention in the body of certain products of urinary or renal origin. Strictly speaking, uremia means simply blood containing excrementitious urinary substances.

Although most common in Bright's disease, uremia may arise also in other diseases, as in gout (gouty kidney), scarlet fever (scarlatinal nephritis), typhus fever, yellow fever, and cholera, in which the kidneys and blood may be seriously affected. Kidneys which, on account of marked structural changes, fail to eliminate the normal quantity of urates and solid constituents are directly or indirectly responsible for an association of the morbid conditions known as uremia.

Our present knowledge of the **pathology** and **etiology** of uremia, as of renal edema, is based solely upon theoretic views. The theory that attributes uremic symptoms to the retention of the excretory products appears to have the strongest proofs to support it; but the positive nature of these substances, or which is the most toxic, or whether several are concerned in the causation or not, remains to be determined. Since

the urea and uric acid have been found in increased quantities in the blood of uremic patients, and since these products are diminished in the urine of nephritis, they also were at first supposed to be the cause. Ammonium carbonate, it was alleged by Frerichs, operated in the same manner after it accumulated in the blood in sufficient quantity as a result of the decomposition of the urea by a ferment.

Not only some of the solid urinary constituents accumulate in the blood in uremia, but the water also is only partly eliminated, and its presence in the blood renders the latter hydremic and of lower specific gravity. Notwithstanding the fact that most cases of uremia may be traced to a marked simultaneous diminution in the quantity of urine passed, there remain still certain instances of renal disease in which uremic symptoms appear without any such perceptible diminution. Even more frequent perhaps are those perplexing cases of anuria now and then reported in which no uremic symptoms appear. In the latter instances it is probable that the elimination of products normally excreted by the kidneys may be accomplished through other channels, as by the skin and bowels; in the former it is still likely that the solid urinary constituents are retained, even with an undiminished quantity of water excreted.

Traube's theory of the cause of uremia, particularly of the nervous or cerebral manifestations, was that it is an acute edema of the brain—local or general—with cerebral anemia. This would seem to explain certain cases of nephritis, as already mentioned, in which a fair amount of urine and solid constituents are passed; also cases of anuria due to urethral obstruction in which no uremic symptoms appear; and certain cerebral disturbances. But with our present knowledge of the chemico-pathology and of the clinical cause of the uremia of nephritis in all its forms there is, I think, no doubt that most cases are caused by the toxemia produced by the retention of the mass of excrementitious substances due to an abatement of the renal functions.

Delafield, however, attributes the sudden violent motor symptoms of acute uremia to a contraction of the arteries from some unknown cause other than blood-contamination.

The **symptoms** of uremia may be either acute or chronic in onset, severity, and course. In **acute uremia** the *severest nervous symptoms* come on suddenly; they last but a comparatively short time, and terminate fatally, with convulsions and coma, dyspnea, feeble cardiac action and pulse, fever, and pulmonary edema. These acute symptoms, however, are not infrequently preceded by mild *uremic prodromes*, as headache, somnolence, nausea, malaise, slight dyspnea, and uneasiness.

**Chronic uremia** is characterized by the *absence* of the marked symptoms referred to above, the milder manifestations alone appearing and lasting over a considerable length of time. Here the general prostration, the feeble cardiac and arterial states, the occasional stupor and delirium, transient dimness of vision, anorexia and nausea, irregularly hurried breathings, and muscular twitchings, indicate the grave condition of the patient. To gain a more thorough knowledge of this interesting and serious complication of renal disease a divisional study of the symptomatology is necessary.

**Cerebral Symptoms.**—These vary from a slight headache, tremors, and



the restlessness of anxiety to the most violent maniacal delirium and convulsions; from somnolence, low muttering, and mental stupor to profound coma; and from slight visual disturbances to complete amaurosis. The onset of a noisy delirium, and less commonly of a marked mania, is often abrupt, and may be the first manifestation of Bright's disease in an individual. Delusional insanity (*folie Brightique*) is seen in a few cases. Bischoff has observed only two cases of purely uremic psychoses among 3000 cases of insanity, and believes that a neuropathic tendency, chronic alcoholism, and pregnancy are the most important predisposing causes. Melancholia and the delusion of persecution, with suicidal and homicidal tendencies, may thus occur. The most characteristic symptom of uremia, however, is the convulsion (uremic eclampsia). *Uremic convulsions* are epileptiform in type, although they may be either unilateral or local—of the Jacksonian form of epilepsy. They are supposed to be due to a local or general edema of the brain, and are probably allied to the *apoplexia serosa* of early writers (Osler). The convulsions of uremia may come on suddenly or may be preceded by headache, vertigo, dropsy, nausea, and vomiting. As in the epileptiform convulsion, after the early tonic rigidity there may follow at short intervals the clonic spasm, with cyanosis, fever, and contracted arteries, and the intervening periods of unconsciousness, shallow or noisy respiration, and slow, hard pulse. *Coma* may come on gradually as well as during the convulsive attacks. It may be preceded by headache, apathy, and insomnia, and continue progressively to deepen for a long time. A *typhoid state* not infrequently accompanies uremic coma. The temperature is usually lowered, and moderate dilatation or contraction of the pupils may be evidenced.

*Uremic Amaurosis*.—Blindness may follow uremic convulsions, or, rarely, it may come on without motor disturbances. It is of purely centric origin (the cortex of the occipital lobe), and its duration is short, lasting but a few days in most instances. *Uremic deafness*, which is probably also of centric origin, is a less common manifestation. Other nervous phenomena, as hemiplegia, monoplegia (from cerebral or spinal congestion or edema), contractures, aphasia, pruritus, paresthesiæ, and cramps in the calf-muscles are not so frequent in occurrence.

*Circulatory Disturbances*.—The pulse is moderately slow, tense, and full in uremia, but with the onset of acute and severe symptoms, as convulsions, it usually becomes accelerated, small, and feeble. The heart's action is labored and feeble.

*Respiratory Symptoms*.—Renal dyspnea, which is sometimes called "uremic" or "renal asthma," is a marked, rather constant, and often an early symptom of uremia. The respirations are deep and often stertorous in coma, or they may be irregular, accelerated, and shallow, sometimes assuming the Cheyne-Stokes type. Dyspneic attacks are especially apt to occur at night. In chronic uremia slight dyspnea may be continuous for a long time. Again, alternating paroxysmal exacerbations may arise. The uremic dyspnea is probably due in most cases to the toxemia affecting the respiratory nervous centers. It may, however, be the result of cardiac weakness or of dropsy or pulmonary edema.

*Gastro-intestinal Symptoms*.—Uremic stomatitis is generally seen. The breath is foul, the tongue, lips, and gums are red, swollen, and pain-

ful, and the saliva is increased. Uremic vomiting is also usually of centric origin, though it may be provoked by the irritation of the gastric mucosa, caused by the vicarious elimination of the urea and the decomposition of the latter into irritating ammonium carbonate. The vomiting may come on suddenly and be persistent. Uncontrollable hiccough and sometimes uremic diarrhea may be associated. The irritant action of the ammonium carbonate on the intestinal mucous membrane may produce a catarrhal or diphtheritic inflammation, and ulceration even (Grawitz). Uremic diarrhea may also exist apart from any marked gastric disturbances.

**General Symptoms.**—The *skin* of the face is usually pale in uremic coma. *Urea* may be excreted by the sweat-glands, and may be seen as minute glistening crystals in some of the cutaneous furrows after the evaporation of a free sweat. The skin is often harsh and dry, as in chronic interstitial nephritis. Uremic pruritus is probably the result of the peripheral irritation of the cutaneous nerves by crystals of urea. The *temperature* is generally lowered, but uremic fever frequently accompanies the convulsions or they may be preceded by “uremic chills.” In some cases the temperature rises to  $105^{\circ}$ – $107^{\circ}$  F. ( $40.5^{\circ}$ – $41.6^{\circ}$  C.) just before death, whilst in other cases, characterized by a profound and lasting coma that deepens into collapse, the temperature may be so low as  $91^{\circ}$  or  $93^{\circ}$  F. ( $32.7^{\circ}$ – $33.8^{\circ}$  C.).

There is not infrequently an *ammoniacal odor* about a uremic patient. The *urine* is diminished in quantity, is generally highly albuminous, and deficient in urea. A previous dropsy is sometimes markedly reduced upon the appearance of acute uremic symptoms.

**Duration and Prognosis.**—Acute uremia is manifested by coma and convulsions, seldom lasting more than a few days. Chronic uremia, in which milder nervous symptoms, nausea and vomiting, and dyspnea are more prominent, may persist, however, for many weeks. While a grave condition, uremia, even in its most acute and violent forms, is not at once necessarily fatal, for under proper treatment—as by venesection, for instance, followed by judicious hygienic measures—life may be considerably prolonged. Sooner or later, however, barring a possible death from some intercurrent affection, a fatal result is inevitable.

**Diagnosis.**—Uremia may be recognized by the history, the marked arterial tension, and the accentuated second sound of the heart; also by the albuminuria (the urine has to be withdrawn), the temperature, and the odor of the breath. The presence of dropsy in some cases is a valuable indication of the nephritic origin of uremic manifestations.

**Differential Diagnosis.**—Uremic unconsciousness coming on suddenly, as in chronic interstitial nephritis, may simulate *alcoholism*, *cerebral hemorrhage* (*apoplexy*), *cerebral tumor*, or *meningitis*. The points of dissimilarity between the first two conditions and uremia are here tabulated (Herrick):

CEREBRAL HEMORRHAGE.	ALCOHOLIC NARCOSIS.	UREMIA.
Pupils unequal or dilated.	Pupils contracted or dilated; eyes injected.	Pupils generally dilated; albuminuric retinitis.
Stertorous, puffy breathing, and flapping cheek.	No stertorous breathing.	Sharp, hissing stertor.
No odor.	Odor of alcohol.	No odor, unless urinous.

CEREBRAL HEMORRHAGE.	ALCOHOLIC NARCOSIS.	UREMIA.
Paralysis ; hemiplegia.	No paralysis, usually.	No paralysis.
Unconsciousness absolute.	May be aroused.	May or may not be aroused.
Pulse slow and strong or irregular ; arteries often atheromatous.	Pulse frequent and feeble.	Pulse at first strong, later weak and rapid ; tension strong ; arterio-sclerosis.
Coma sudden and deep.	Coma gradual.	Coma gradual or sudden.
Convulsions late ; may be unilateral.	No convulsions.	Preceded by general convulsions, headache, etc.
Urine generally negative.	Urine generally negative.	Urine albuminous.
Apoplectic habit ; heart may show hypertrophy.	Red face and nose, heart often weak, dilated, myocarditic.	Edema and pallor ; heart hypertrophied.

In *meningitis* the mode of onset, the rigidity of the neck, incoherence or mild delirium, photophobia, and pronounced fever point to the distinction.

Uremic coma must also be differentiated from *opium-poisoning* and *diabetic coma*. Chronic uremia must not be confounded with the asthenic state of *typhoid fever* and *acute miliary tuberculosis*. In *opium-poisoning* the pupils are contracted and do not respond to light. Again, in *opium-poisoning* the respirations are slow, deep, and full, and the patient may answer rationally when aroused. In uremic coma, it will be remembered, consciousness is abolished. In *diabetic coma* the history must be learned, the harsh, dry skin and emaciation noted, and especially are the ethereal odor and the Burgundy-red reaction of the urine (acetone) with the tincture of the chlorid of iron to be observed ; sugar is also present.

The **prognosis** is grave, but guarded ; it is even favorable in many cases, so far as immediate results are concerned.

**Treatment.**—This will be detailed in the discussion of the various forms of nephritis. Suffice it to say that the supreme indication is the prompt elimination of the poisons in the blood. When diaphoresis and catharsis fail either in promptness or efficiency, venesection should be employed ; the latter measure is also probably the most reliable in urgent cases of uremic convulsions or coma. The counter-injection (intravenous) of normal salt solution may be indicated in cases of profound weakness threatening collapse.

Bozzoli recommends the subcutaneous injection of sterilized serum because of the gratifying results secured in a number of cases of uremia.

## AMYLOID KIDNEY.

**Definition.**—Amyloid (waxy or lardaceous) degeneration of the kidneys ; it is usually coexistent with a similar degeneration of other viscera.

**Pathology.**—Macroscopically, the amyloid kidney appears pale, greenish or yellowish-white, firm, and uniformly enlarged, and the surface is smooth, glistening, and often mottled, owing to the prominence of the stellate veins. On section a homogeneous, anemic, or “bacon-like” surface presents itself, particularly in the cortical region. The cortex is wider than normal ; the pyramids may be red in color and slightly



infiltrated; and the glomeruli may show an infiltration by the glistening, translucent amyloid (albuminoid) material. On the application of Lugol's solution of iodine to the amyloid areas a mahogany-red color is produced. Brushing over the amyloid substance with a solution of iodine, and then with dilute sulphuric acid, gives a blue or violet tint. Similarly used, a 1 per cent. solution of methyl-violet strikes a red color. The capsule of the kidney is thickened, though not always adherent.

Microscopically, the amyloid change is generally found in the early stages to affect the walls of the capillaries of the Malpighian tufts. The walls are swollen with the homogeneous material and the vessel-lumen is diminished or obliterated. The straight uriniferous tubules are also infiltrated later perhaps, the deposit occurring primarily in the *membrana propriae*. A diffuse nephritis is nearly always an associated condition. The tubules generally contain hyaline casts. Fatty degeneration of the epithelium, glomerulites or waxy glomeruli, and a thickening of Bowman's capsule are common in markedly amyloid kidneys. In advanced cases most of the secretory structure becomes atrophied. Amyloid infiltration of the smaller granular kidney is less common than of the large white kidney, with intense parenchymatous changes.

Hypertrophy of the heart is not always present in amyloid disease of the kidneys. Amyloid infiltration of other organs, however, as of the liver and spleen, is usually associated with waxy kidneys.

**Etiology.**—The causes of amyloid kidney are those of the amyloid change affecting (either simultaneously or nearly so) other organs, as the spleen, liver, and intestines.

Commonly, amyloid disease is marked also in the other solid organs named above; it is secondary to wasting diseases, cachexiae, and the like. Perhaps the most frequent cause of the waxy kidney is tuberculosis, especially of the lungs ("chronic ulcerative phthisis"): tuberculosis of the intestines also is often associated and aggravates the amyloid infiltration. Next in order are the prolonged suppurations, particularly of the bones, as in osteitis of the vertebrae and hips (usually tuberculous). Chronic empyema, intestinal ulcers, vesico-vaginal fistulae, and other purulent affections, chronic in nature also, have the same etiologic effect.

Amyloid kidney is often present in syphilis, especially in the tertiary stage, when ulceration of the mucous surfaces and of the bones is present. Rarely, gout, malaria, leukemia, cancer, and chronic valvular endocarditis with insufficiency seem to produce amyloid disease.

**Symptoms.**—These vary greatly according to the extent to which the amyloid degeneration has encroached upon the normal kidney-structure, and may be overshadowed partially or completely by those of the dominant causal affection.

The *urine* is pale yellow, clear, and variable in quantity, and the amount passed in twenty-four hours is sometimes normal or may be slightly diminished. More frequently, perhaps, it is increased, and especially in marked or advanced cases. The specific gravity is apt to be low (1015–1005), and there is seldom any sediment.

*Serum-albumin* and *globulin* may both be present in the urine; but a highly significant condition, and one that is seemingly diagnostic, is the high proportion of globulin as compared with the serum-albumin (Salkowski, Senator). *Tube-casts* may be found, but their presence may be

only temporary; they are usually wide hyaline or fatty and granular, and are few in number (Fig. 66). The amyloid reaction may be elicited with the hyaline casts; symptoms referable to the kidney are often absent in comparison with those of the nephritides. *Dropsy* is not invariably present, and when present is but moderate in degree and generally in the legs only. It is proportionately prominent with the increase in the anemia, circulatory depression, and wasting of flesh and strength. The latter manifestations, constituting a cachectic appearance, are quite commonly observed in amyloid kidney.

The associated enlargement and the firm, sharp outlines of the liver and spleen are of diagnostic significance. *Marked diarrhea* may be due to coexisting amyloid infiltration of the intestines or to tuberculous intestinal ulcers, and is often seen in advanced cases.

**Diagnosis.**—This can seldom be made upon the urinary manifestations alone. Important and often necessary adjuncts are the histories of causation and of the associated symptoms and physical signs. Thus, there will be evidenced in most cases tuberculosis, chronic bone-suppurations, or syphilis, while coexisting hepatic and splenic enlargements, wasting, and cachexia are usually present. In any of the diseased conditions mentioned amyloid kidney may be diagnosed with reasonable certainty upon the development of an increased quantity of pale clear urine of low specific gravity and containing a large amount of albumin, or even with slight albuminuria.

From *parenchymatous nephritis* amyloid kidney is to be differentiated by the history, by the more marked and generally distributed dropsy, and by the albuminuric retinitis that characterize the former. In *chronic interstitial nephritis* there are less marked albuminuria and dropsy, and there are present arterio-sclerosis, cardiac hypertrophy, and a pronounced tendency toward uremic symptoms.

**Prognosis.**—This varies with the cause. Incipient bone-disease or tuberculosis, with only slight evidences of amyloid change in the kidneys, may be controlled. As a rule, however, the structural alterations are so far advanced, and the constitutional powers of resistance so much enervated, before the amyloid infiltration can be distinctly apprehended that in the majority of instances the prognosis is entirely unfavorable. In decided cases death ensues in from several weeks to as many months.

**Treatment.**—This also depends upon the causal affection. Hygienic and dietetic measures are always useful, however, with a view to improving the general nutrition. The iodid of iron has been recommended as an alterative, and easily assimilable and palatable fats and tonics may also be tried. Tuberculous cases require creasote or allied preparations; syphilitics require mercurials and iodids; while malarial subjects do best under the systematic use of arsenic, iron, and quinin.

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## NEPHROLITHIASIS.

(*Renal Calculi; Pyelitis Calculosa; Renal Colic; Gravel.*)

**Definition.**—A condition characterized by the formation of fine or coarse concretions in the kidney-substance or in the renal pelvis by the precipitation of certain of the solid urinary constituents.

**Varieties.**—According to their size, renal concretions are variously termed—(1) **Renal sand**, of which the particles are fine and pulverized; (2) **Renal gravel**, consisting of coarse grains or even of pea-sized concretions; (3) **Renal stone, or calculus**, when larger masses than the preceding exist, either more or less rounded or as stony casts or moulds of the pelvis of the kidney, its infundibula, and calyces (*dendritic* or *coral calculi*).

According to their composition, the chemical varieties of renal concretions are—(1) *Uric-acid* calculi, the most frequent in occurrence. Urates are often associated in the calculus with uric acid, thus producing stratification. These concretions may occur as sand, gravel, or large stones; they are usually quite hard, reddish-brown or black in color, and have a smooth though irregularly-shaped surface. The fracture is crystalline, and in the larger calculi often shows the alternating layers of uric acid and ammonium urate. Pure uratic stones may occur in children.

(2) *Calcium-oxalate* concretions occur more rarely in the kidney. They constitute the so-called “mulberry calculi,” from a fancied resemblance to the mulberry, owing to their dark-brown or black color and very irregular and nodulated or prickly appearance. They are also quite dense; lamination, however, is not common, although they are sometimes formed about a uric-acid nucleus.

(3) *Phosphatic* calculi of the kidney are still less common than the oxalate, but they are more common in the bladder. They may consist of calcic phosphate or ammonio-magnesian phosphate, and may possibly be associated with calcic carbonate. Phosphatic salts are most often deposited secondarily about uric-acid or oxalate calculi in the alkaline urine of a cystitis set up by the irritation of the true renal stones. Phosphatic calculi are grayish-white in color and are comparatively soft.

(4) Renal stones composed of *cystin*, *xanthin*, *carbonate of lime*, *fatty or saponaceous matters* (urostealith), *indigo*, and *fibrin*, though of extreme rarity, have been occasionally reported. Cystin calculi have a pale-yellow color and a waxy luster.

**Pathology.**—The anatomic changes of the kidney vary with the degree and persistence of the irritation, the size of the calculi, and their passage or retention. Sometimes numerous granular and pea-sized concretions are found in the renal pelvis, with desquamated epithelium and a turbid urine. Interesting cases are those in which a dendritic stone occupies a great portion of the atrophied kidney-substance, as well as the entire pelvis of the organ. In one of my own patients the left kidney was, apparently, nearly twice the normal size, owing to the presence of a large coral-calculus (uric acid and urates), connected by an isthmus with a rounded stone in the inferior portion quite as large as a large walnut. The pelvis of the right kidney also contained a dendritic calculus.

**Secondary Lesions.**—Perhaps the most usual result of renal concretions is a pyelitis: this may be simple catarrhal, diphtheritic, or purulent, with or without hemorrhages, depending upon the intensity of the mechanical irritation. A pyelo-nephritis may follow in severe cases, as may even a general suppuration (pyonephrosis) or perinephric abscess and perforations. Renal pus-cavities are sometimes found *postmortem* containing numerous small stones. Hydronephrosis is another important pathologic sequel, in which the cause is to be attributed to the blocking of the ureter by an erstwhile passing stone or by the closing of the aperture of



a ureter from within the pelvis. Pressure-necrosis and perforation may thus be induced. Owing to the prolonged pressure of a dendritic calculus, there is commonly a distinct and marked atrophy of the renal parenchyma, resulting in chronic diffuse nephritis with little or no exudation.

**Etiology.**—The definite causation and the exact manner of formation of renal concretions are still unestablished. We may infer not a little, however, with some good reason, since the *predisposing causes* are rather distinct. Thus, in children and in advanced life (before 15 and after 50 years of age—Purdy) the occurrence of calculi is most common, the uratic variety being most frequent in the former and the uric acid in the latter. Men are subject to nephrolithiasis more often than are women. The uric- or lithic-acid state (lithemia), gout, and the various influences that induce these conditions, as an excessive meat (proteid) diet or a sedentary life, seem to predispose to stone. Heredity, I believe, plays a prominent part in many cases.

Broadly speaking, any habit of the system that encourages the precipitation of insoluble abnormal ingredients or of normal ingredients in excess, owing to chemical changes in the urine, tends to the formation of calculi. It should be stated, however, that the *primary causes* of calculus-formation is the presence of some substance in the urinary tract that affords a nucleus about which the successive layers of crystals may deposit and adhere, such as bits of mucus, colloid material, epithelial shreds, parasitic ova, bacteria, blood-clots, and tube-casts.

It is generally believed that the requisite conditions for the formation of a uric-acid renal calculus are—a highly-acid urine, an excess of uric acid, a low percentage of salines, and deficiency of the normal urinary coloring-matters.

**Symptoms.**—These may be slight, progressive, and chronic, or they may be intensely acute and comparatively short in duration, though subject to repetition—*i. e. renal colic*. It is not unusual for patients to pass uric-acid sand and gravel for years without much complaint. A sudden blocking of a ureter, however, or a slowly-passing stone of distending dimensions produces great agony at times. A smooth, snugly-fitting dendritic calculus in the pelvis may not cause any symptoms for years until the destruction of tissue by its weight and mechanical irritation ensues; there is then a progressive failure of health, a constantly increasing pain in the back, *occasional hematuria*, tenderness on pressure over the diseased kidney, both anteriorly (deep) and posteriorly, and finally *uremia* and death.

The characteristic symptoms of stone in the kidney appear as an attack of *renal colic*. This happens when a calculus in its passage down the ureter acts as a mechanical irritant, or when it is caught and stopped in the passage. The large “gravel” or pea-sized and more or less rough stones usually cause the attack, which comes on, as a rule, quite suddenly, although it may be preceded by a chill and some general uneasiness or by slight pain in the region of the kidney. It may be excited by a sudden muscular effort. The pain is tearing in character, and rapidly reaches an agonizing maximum of severity, starting from the lumbar region and extending down along the ureter into the groin, and often into the testicle and inner side of the thigh. The paroxysm may appear in the form of a diffuse abdominal and lumbar pain in some instances. There

is local tenderness on pressure, and nausea and repeated vomitings are frequent. The patient is often collapsed, and perspiration, a rapid, small, and feeble pulse, trembling, anxiety, bodily twistings about, convulsions even, and syncope may ensue. There may be moderate fever. The *urine* is scanty or may be suppressed for a time, and is often bloody. Frequent and painful attempts at urination are made, with the passage of but a few drops at a time, owing perhaps, in part at least, to a reflex spasm of the vesical sphincter (vesical tenesmus). The presence of pus and of pelvic epithelium in the urine indicates a pyelitis. When a large quantity of clear urine is passed, as sometimes happens, it may be looked upon as having come from a healthy kidney.

The *paroxysm* of renal colic ends when the impacted stone passes out of the ureter. This may occur within a few hours or it may take several days; in instances of the latter type the attacks of renal colic may be intermittent.

Recovery is not always complete immediately upon the evacuation of the stone. The previously retracted testicle may be painful and swollen for a little while, and there are apt to be aching and soreness over the affected kidney and ureter.

In certain severe cases of mechanical irritation the symptoms of pyelitis, pyelo-nephritis with abscess, or hydronephrosis may be superadded. Anuria and uremia result from simultaneous obstructive suppression of the urine upon both sides.

Nephrolithiasis as a *chronic affection* may exist for many years, with recurring paroxysms of renal colic. I observed a case for five years that had extended over a period of thirty years, until it finally came to necropsy. Between the attacks of colic the patient may be entirely comfortable, save perhaps an occasional burning in the urethra on micturition, owing to a highly-concentrated, acid urine or to the passage of minute uric-acid granules. There are apt to be pain and tenderness over a kidney containing a large imbedded stone. A smoky-hued urine, due to slight hematuria, is also sometimes present in long-standing cases of renal calculus, particularly after exertion.

A *renal intermittent* fever, simulating malarial paroxysms, may occur in nephrolithiasis, and is analogous to the hepatic intermittent fever of cholelithiasis.

*Pyelitis*—simple or purulent—with late involvement of the kidney-parenchyma (pyelo-nephritis) is a frequent concomitant of chronic nephrolithiasis. The presence of pus in the urine is constant, with an absence of renal epithelium in cases of an abscess-cavity of the kidney. In ordinary pyelitis the pyuria is often intermittent.

The general health of patients with nephrolithiasis is, as a rule, remarkably good. Anorexia is not only seldom present, but such persons are habitually free and good livers. Persistent headaches with nausea, however, should warn one of uremia. Splenic and hepatic enlargement may be found with prolonged suppurative pyelo-nephritis, indicating amyloid disease.

**Diagnosis.**—This resolves itself into a study of the diagnostic characters of (*a*) the attacks of renal colic, (*b*) of the underlying systemic condition in general, and (*c*) the renal condition in particular that renders these attacks possible. The latter can be discovered only by a careful

and continuous study of the clinical history and urinary manifestations as outlined in previous paragraphs.

Nephrolithiasis may be positively diagnosed in a case in which, after sudden, agonizing, colicky pain, referred to either lumbar region and radiating down the ureteral course to the testicle, a concretion is found to have passed with the urine. It is therefore necessary in a suspected case of renal colic to pour the urine through a fine sieve as soon as passed. The more recent improvements in the operative technic for producing the Roentgen rays enable us to detect renal calculi with considerable accuracy as to their number, size, and relative position.

**Differential Diagnosis.**—Renal colic must not be taken for *biliary or intestinal colic*. The antecedent history is of great value in arriving at a diagnosis. In biliary colic there may be jaundice, and pain referred to the upper rather than to the lower abdominal zone, both of which symptoms are absent in renal colic; while in the latter the disturbance of micturition and the character of the urine, especially the hematuria, are characteristic.

In intestinal colic the griping pain is usually most intense in the umbilical region, is often relieved by pressure, and is associated with tympanites and constipation; it has usually a dietetic origin, while the renal and urinary symptoms are absent. The exclusion of *lumbodynia* and *lumbo-abdominal neuralgia* is not so difficult. The differentiation of the varieties of calculi from the symptoms is not positive. It has been suggested, however, that the oxalate stones usually cause the sharpest pains and the hematuria. Right-sided ureteral pain felt over the lower abdominal region may be confounded at first with *appendiceal colic*. Musser has found the pain of renal colic to be more paroxysmal and less uniform in location than in the latter however. Early *renal tuberculosis* (*vide*) with its hematuria and pyuria must be differentiated from renal calculus also.

**Prognosis.**—This should always be guarded, owing to the possible dangers and complications that frequently attend nephrolithiasis in all of its forms. Thus the passage of gravel without marked symptoms tends to persist or recur—in both events an unfavorable tendency, since subsequent formations are apt to be larger and cause serious symptoms. An attack of renal colic may itself be fatal. Large latent calculi (dendritic), of long standing, are nearly always incurable, and in most instances lead to such grave complications as pyelo-nephritis, pyo- and hydronephrosis, perinephric abscess, and uremia.

**Treatment.**—Paroxysms of renal colic call for prompt relief. This is best afforded by hypodermic injections of morphin and atropin, coupled with hot baths or fomentations applied to the loins. The free use of hot drinks, as lemonade, soda, or plain water, is also helpful in promoting the passage of the stone. Cases of excessive suffering require the inhalation of chloroform.

The treatment of the nephrolithiasis without or between attacks of renal colic is most important. First to be considered are the **hygienic** and **dietetic** measures. for in mild and uncomplicated cases much can be done to prevent the aggravation of the disorder, and at least the formation of larger concretions may be delayed. When the tendency is to uric-acid gravel (the commonest variety), the patient should live a reg-



ular, calm, steady, and temperate life. Exercise should be so managed that it may be taken rather moderately in the open air, and with a view to preventing additional weight in persons of fair nutrition and to promoting a reduction of weight in the obese. In short, the exercise should be sufficient to thoroughly use up all nitrogenous food, so that the formation and elimination of urea may be increased to normal and the quantity of uric acid diminished. Hence I would strongly advise a clinical study of the percentage of urea in the urine (*vide* p. 957).

Over-indulgence in food, particularly in red meats (liver, sweetbread, and similar nuclear food), should be prohibited, owing to the ready formation of uric acid from the latter. Alcohol should be taken seldom, or, better, not at all. On the other hand, since the urine is apt to be scanty and highly acid, the patient should be encouraged to drink freely of plain and alkaline waters, artificial and natural. The value of various pure spring-waters as diluents is undoubted, the Buffalo, Londonderry, and Otterburn Lithia, the Saratoga, Bedford, and Poland waters, all being distinguished for their purity. More marked and more generally useful for their alkalinity are the Carlsbad, Vichy, and carbonated waters. In cases characterized by occasional hematuria the Rock-bridge alum-water may be tried. Plain soda-water and lemonade may be used as adjuvants.

The **medicinal** treatment of nephrolithiasis is aimed to secure a solvent and disintegrating action upon the stones; it is symptomatic. It is extremely doubtful whether stones once formed in the pelvis of the kidney and remaining there are ever dissolved, though certain drugs would seem to have had an eroding effect in some instances, and they are to be recommended as useful in preventing the formation of new deposits. Lithium citrate or carbonate in 5-grain (0.324) doses in tablet form, three or four times daily, has been generally employed for the purpose. Sodium phosphate and the vegetable salts of potash, as the citrate, acetate, and tartrate, are useful. Much water, especially the carbonated, should be drunk, along with doses of the above, in order to facilitate the solvent action, and in this way relieve, in a measure, the local distress and pain. Recently piperazin has been brought forward as an uric-acid-calculus solvent by some clinicians, and that it has an action such as is claimed for it has been proved beyond a doubt in certain cases. Whilst it deserves a further trial in nephrolithiasis, it is too much, however, to expect to look for positive and successful results in every case. It is prescribed usually in 5-grain (0.324) tablets three or four times daily, with much water.

Recently, Van Noorden and Strause have recommended calcium carbonate (gr. x-xv—0.648–0.972—or more thrice daily). The theory is that the calcium unites with the acid phosphates in the intestines, and thus reduces the deuterophosphates in the urine, leaving the protophosphates to dissolve the uric acid. They report excellent clinical results.

The reaction of the urine must be tested at stated intervals and kept faintly acid. Should the urine become alkaline, the alkaline treatment must be suspended for a while, or a secondary deposit of phosphates about the uric-acid stone may be induced. Nagging lumbar pains may be relieved by occasional doses of such analgesics as phenacetin, belladonna, hyoscyamus, codein, and indirectly by the sweet spirits of niter, buchu,

and uva ursi. Renal hemorrhage may be controlled effectually by the use of the fluid extract of ergot, or by alum in 10- or 15-grain (0.648 or 0.972) doses, or by gallic acid in 20- or 30-grain (1.29–1.94) doses.

Efforts to acidify the urine are indicated when the calculus happens to be composed of phosphates or of calcium carbonate. This is more difficult of accomplishment than when it is necessary to reduce the acidity. Saccharin in 2- or 3-grain (0.129–0.194), and benzoic and boric acids in 5- to 15-grain (0.324–0.972) doses, in capsules, seem to be most useful for this purpose. It is claimed for calcium carbonate, again, that it diminishes the phosphates without making the urine alkaline.

The question of surgical interference must be decided in not a few cases; thus, it may be briefly stated that in protracted and obstinate cases of calculous renal disorder, with persistent local pain, a gradually decreasing capacity for work, and evidences of severe pyelitis, pyelonephritis, or, worse, of perinephric abscess, the surgeon must operate. In the simplest cases a nephrotomy or nephro-lithotomy may be performed and the stone removed. Where the renal structure is much damaged it may be necessary to do a nephrectomy. To avoid the increased perils of the latter operation, however, it were better that a nephrotomy were done as early as consistent with the diagnosis of incarcerated pelvic stone and the condition of the patient.

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### ACUTE NEPHRITIS.

(*Acute Bright's Disease; Acute Diffuse Nephritis; Acute Parenchymatous Nephritis; Exudative, Catarrhal, Tubal, Desquamative, and Glomerulo-nephritis of Acute Course.*)

**Definition.**—An acute inflammation of the kidneys, more or less diffuse in nature. It may be either of a mild, severe, or grave character. Delafield describes three varieties of acute renal inflammation under the common synonym of acute Bright's disease, as follows: (1) *acute degeneration of the kidneys*, (2) *acute exudative nephritis*, and (3) *acute productive nephritis*. This division is of etiologic and pathologic importance and interest, rather than of clinical necessity or practical value.

**Pathology.**—The anatomic changes in, and the appearances of, the kidneys vary considerably in different cases according to the degree of involvement. From the very mild to the gravest cases of nephritis there is an intermediate series of continuously more marked pathologic changes in the renal tissues. These depend greatly on the amount of poisonous material circulating in the kidneys and eliminated by them, as well as upon the intensity and duration of its noxious action.

In the mildest cases the *macroscopic* appearances of the kidneys may present nothing distinctly abnormal. As a rule, however, the organs are slightly enlarged, swollen, and somewhat softened. These conditions are more evident when the interstitial exudation is abundant and when inflammatory edema is evident. The kidneys may be reddened and congested and appear bloody on section, or they may be pale and mottled. In examples of the former, hemorrhages may be formed beneath the cap-

sule (*acute hemorrhagic nephritis*), though it is more common to see red patches of hyperemia alternating with opaque, whitish portions on both the outer and cut-surfaces of the kidneys. The cortex especially is swollen, turbid, and pale, or slightly congested in the mildest cases, and is deeply mottled (red and pale glomeruli) or hyperemic in severe instances. The pyramids usually show an intense redness. The surfaces are smooth and the capsule non-adherent.

*Microscopically*, alterations may be discovered that are not visible to the naked eye in the very mild cases referred to above. There is simply a cloudy swelling or a granular (parenchymatous) degeneration of the epithelium of the Malpighian tufts, Bowman's capsule, and of the cortical uriniferous tubules. This is not true acute nephritis, however, in the absence of exudative changes in the interstitial tissue. The acute parenchymatous degeneration may be almost exclusively limited to the glomeruli, as in some cases of scarlatina, and hence the term *glomerulo-nephritis*. The cells are swollen, opaque, and irregular in shape, while the cell-contents are granular (albuminoid or fatty). A further advance in the process is seen in cellular coagulation-necrosis or disintegration, desquamation of the cells, and hyaline degeneration of masses of them in the tubules. Acute degenerative changes are frequently found in the acute infectious diseases or when inorganic poisons have been introduced into the body. In phosphorus-poisoning actual fatty degeneration of the epithelium may be found, this either proceeding from the cloudy swelling or developing independently. A rapid necrosis of cells is also met with in severe cases.

*True acute nephritis* is not only characterized by changes of the renal epithelium (the parenchyma), but the inflammatory exudate (serum, leukocytes, and erythrocytes) is found between the tubules. The kidneys show different stages of the process in different portions. In some places there is only a slight cellular infiltration of the intertubular tissues; in others, besides the desquamation of necrotic epithelial cells and the presence of hyaline casts in the tubules, the interstitial tissue is swollen by the coagulated sero-fibrinous exudate, abundant leukocytes, and some red blood-corpuscles. It should be stated that the inflammatory exudate collects also in the Malpighian bodies and tubules. The epithelium lining the latter, especially the convoluted portion, is often flattened, and the tubules themselves may be dilated and choked with degenerated cells, or, more frequently in the straight tubules, with hyaline casts. The white blood-cells that are found infiltrating the stroma of the kidney are not usually equally diffused, but are collected in foci in the cortex.

The glomerular epithelium of the capsule, and especially that covering the outside of the capillaries of the tufts, is swollen and opaque, and the outlines of the individual capillaries are lost. In most cases of diffuse exudative nephritis new epithelium appears, and a restoration of the glomerular function takes place. In the *productive variety* of acute diffuse nephritis, however, according to Delafield, the lesions consisting of a cellular growth in the capsules and of connective tissue around thickened arteries—are more permanent in character from the first, and hence the increased gravity of the disease. In the more intensely acute cases the new tissue between the tubules is largely cellular; in those of a subacute type it is relatively dense and fibrous.



Anasarca and pleural, pericardial, and peritoneal dropsy are also found in those dying of acute Bright's disease. Cerebral edema, meningitis, and lobar pneumonia are to be mentioned as complicating conditions that are sometimes seen *postmortem*.

**Etiology.**—Acute nephritis may occur at any time of life, though it more often makes its appearance before than after middle life. Males are more susceptible than females, and particularly when engaged in occupations requiring exposure to cold and wet. The habitual use of alcoholics also is generally a predisposing cause of acute Bright's disease.

The principal exciting causes of acute diffuse nephritis are the following: (1) *Those acting on the skin*, as cold and dampness, extensive burns, and chronic skin-diseases. In many cases it is difficult to estimate whether the influence of alcoholic intemperance predominates or the exposure incident to it. Thus, acute intoxication from beer-drinking itself may cause an attack of acute nephritis, but it is likely that in most instances the direct exciting cause is cold acting upon the individual in his exposed and maudlin condition. The disease may also be attributed at times to exposure to cold and wet irrespective of alcoholic indulgence. It may be presumed with reason that in such cases there is some inherent or acquired weakness or a susceptibility of the kidneys, rendering them the weak links in the visceral or systemic chain.

(2) *Biologic Toxic Agents.*—These embrace the poisons of the acute infectious diseases, though in the majority of cases scarlet fever is the primary affection. Nephritis may supervene during the height of scarlatina, but more often it occurs in the second or third week of convalescence. Other infectious fevers may also cause acute nephritis (small-pox, typhus, typhoid, relapsing fever, epidemic influenza, cholera, diphtheria, yellow fever, measles, chicken-pox, erysipelas, septicopyemia, acute lobar pneumonia, cerebro-spinal meningitis, dysentery, acute articular rheumatism, and tuberculosis: syphilis is rarely a cause). Acute infectious nephritis may also occur as a primary disorder, and the brunt of the affection may fall either upon the kidney, rather than upon any other part, or upon the organism as a whole, as in the fevers. Manna-berg, among others, has described such cases, and demonstrated streptococci in the urine.

(3) *Chemical Toxic Agents.*—Among the principal irritants of this class are turpentine, cantharides, carbolic and salicylic acids, iodoform, the mineral acids, potassium chlorate, and such inorganic poisons as phosphorus, lead, arsenic, and mercury. The excessive ingestion of highly-acid, spiced, or adulterated foods (as from salicylic acid and lead chromate) may in certain individuals cause acute renal inflammation.

(4) *Pregnancy.*—Here the nephritis (*gravidarum*) comes on in primiparæ, usually in the last months of pregnancy. It is probably caused by renal engorgement due to mechanical pressure, as well as to nutritive disturbances in the kidney, owing to the altered blood-condition.

(5) Finally, latent and insidious *chronic nephritis* may be the cause of an onset of a manifest acute nephritis.

**Symptoms.**—The *onset* varies with the cause of the nephritis, though generally it is rather sudden. Chilliness, nausea and vomiting, pain in the back, and, within twenty-four hours, dropsy, are seen in some cases. Children may be seized with *convulsions* (uremic), and adults are

not less liable to them in severe attacks. Fever may be present, although it is neither constant nor high. The characteristic symptom is the early appearance of *edematous puffiness* of the eyelids and face, with pallor of the skin. Soon (and sometimes at first, even) a swelling is noticed about the ankles and legs, and in marked cases the whole body becomes dropsical, so that pitting on pressure may be observed pretty much all over the bodily surface. In such instances the scrotum and penis or the labia may become enormously distended, the skin having almost a translucent appearance.

*Local symptoms*, as pain and tenderness in the lumbar region, are often wanting and are never marked. There may be a desire to micturate often, accompanied by slight burning and vesical tenesmus, due to the concentrated urine. In very severe dropsy the tense, dry skin, as of the limbs, may be sensitive or even painful to the pressing finger. Movements of the body are often difficult, painful, and distressing in marked anasarca. Intense headache and backache may precede the onset of uremia.

In mild cases the renal condition may be overlooked unless a urinary examination is made. Prostration may be unnoticed, and the patient feel nothing more than a general malaise.

The characteristics of the urine in acute nephritis are all-important. The total *quantity* passed in twenty-four hours is diminished, and may be very scanty, sometimes amounting to not more than from 5 to 25 ounces (150–740 c.c.). Suppression occurs in some cases of toxic origin, when an acute degeneration or necrosis of the renal epithelium takes place, and in the most severe exudative inflammations. The *specific gravity* is increased to 1025 or more early in the case; later it may be as low as 1010 or 1015. The *color* is darker than normally, and is usually smoky-red or reddish-brown, according to the amount of blood passed. If the abnormal morphologic constituents are present in great quantity, a more or less abundant flocculent sediment appears on standing.

Microscopically, some red blood-corpuscles and renal epithelium are found, along with the characteristic *blood*, *epithelial* and *granular tubercasts* (Fig. 61). Chemically the urine is *acid*, and on boiling a *thick, curdy precipitate of albumin* forms. The percentage of the latter by weight varies from  $\frac{1}{4}$  to 1 per cent. The urea and gross solids are diminished.

Other symptoms may develop during the course of acute Bright's disease. If *great general edema* is present, physical signs of hydrothorax, ascites, and hydropericardium may be elicited. The first-mentioned condition is bilateral and causes dyspnea; the second increases the dyspnea by pressing the diaphragm upward; and the last impairs the heart's action. Strümpell describes a form of pneumonia—a “stiff inflammatory edema”—midway between lobar pneumonia and broncho-pneumonia, that sometimes develops in severe cases of acute nephritis. Edema of the conjunctivæ, soft palate, and larynx may also occur. Recently, Lapinsky reported a fatal case of acute parenchymatous nephritis in which severe bilateral sciatic neuritis was associated.

The *pulse* is often hard and tense, and, though slow at first, it may become accelerated later. Cardiac hypertrophy of a slight degree may be detected. The aortic second sound is accentuated. Epistaxis is an

occasional symptom, and subconjunctival hemorrhages are sometimes seen as a result of uremic convulsions that may not have been witnessed. A very constant symptom is the dry, anemic skin. *Uremic manifestations* may ensue at any time during the course of the disease. They appear early in the most severe cases, with intense headache and backache, vomiting, and convulsions.

The clinical course in other cases differs somewhat from the above, which may be considered as the common form resulting from exposure. Acute nephritis *occurring as a complication of the infectious fevers*, except scarlatina, may be characterized by the very slight degree, or even by the absence, of dropsy. Albuminuria, hematuria, anemia, and uremia supervene in the graver affections. In *scarlatinal nephritis*, however, anasarca is common, and slight edema at least is quite constant. During the period of convalescence tube-casts (granular or fatty granular) may be found in the urine (Fig. 62). In mild affections simply a little albumin and a few hyaline casts reveal the parenchymatous degeneration. In cases of *degenerative nephritis* due to mineral poisoning the subsidence of the acute toxic symptoms may be followed by the *typhoid condition*. In the so-called *nephro-typhoid condition*, where typhoid fever begins with pronounced symptoms of acute nephritis, hematuria may be marked. The *nephritis of pregnancy* is usually gradual in its onset. The albumin increases in amount from month to month, and reaches a high percentage during the eighth and ninth months. Some hyaline or faintly granular casts are found (Fig. 64), and erythrocytes rarely appear in the urine. *Danger of eclampsia* is constant until the child is delivered; but recovery is rapid after the birth of the child as a rule.

That variety of *acute (productive) nephritis* in which there is a tendency to the formation of patches or wedges of fibrous tissue is characterized by higher fever, by cerebral and circulatory disturbances of a typhoid nature, and by anemia, dropsy, and a highly-albuminous urine, even though blood may be absent and casts may be few. The dropsy is most apparent in the legs. Dyspnea, vomiting, diarrhea, and a progressive and rapid loss of flesh and strength ensue until convulsions or coma, sometimes preceded by acute maniacal excitement, end in death. Milder cases, lasting from two to four weeks, apparently get well, albumin and casts persisting, however, until, after an interval of weeks or months, another and similar attack occurs. In short, the first acute attack is liable to chronic repetition until a fatal one takes place.

**Diagnosis.**—The condition cannot be overlooked when the urine is carefully examined both chemically and microscopically. The dreaded *eclampsia gravidarum* can, however, be recognized only by repeated urinary examination, especially during the last months of pregnancy. Acute Bright's disease should be suspected, and the urine examined in every case showing pallor of the skin and puffy eyelids, whether general prostration of the health is apparent or not. The characteristic symptoms of acute exudative nephritis, as commonly seen when the condition is due to cold or occurs in scarlet fever, are the following: headache, restlessness, muscular twitching, nausea and vomiting, a tense pulse, moderate fever, dropsy, and anemia. Tube-casts and albuminuria are constant. It should be borne in mind that slight albuminuria occurring in the course of pregnancy or during any of the fevers, *without casts*, is





FIG. 61.



FIG. 62.



FIG. 63.



FIG. 64.



FIG. 65.



FIG. 66.

FIG. 61.—A. G., aged fifteen, male, suffering from acute nephritis. Urine showing granular casts (Queen obj.  $\frac{3}{8}$ ; eye-piece ij.).

FIG. 62.—C. A., aged nine, male. Scarlatinal nephritis, third week of convalescence. Urine showing granular casts (Queen obj.  $\frac{3}{8}$ ; eye-piece ij.).

FIG. 63.—J. D., aged fifty-four, male, suffering from cancer of the common duct and head of the pancreas. Urine showing bile-stained casts (Queen obj.  $\frac{3}{8}$ ; eye-piece iv.).

FIG. 64.—B. J., aged twenty-two, female, suffering from puerperal eclampsia. Urine showing large, finely granular casts (Queen obj.  $\frac{3}{8}$ ; eye-piece ij.).

FIG. 65.—S. A., aged fifty-eight, male. Urine showing granular and fatty casts; post-mortem showed chronic parenchymatous nephritis (Queen obj.  $\frac{3}{8}$ ; eye-piece iv.).

FIG. 66.—C. C., aged forty-two, female, suffering from septicopyemia with amyloid kidney. Urine showing epithelial and (so-called) amyloid casts (Queen obj.  $\frac{3}{8}$ ; eye-piece ij.).

[L. Napoleon Boston.]



not a true nephritis, although the latter may be a more or less remote consequence of the glandular degeneration of the renal epithelium associated with the febrile albuminuria. In addition to the presence of albumin and hyaline and cell-casts, however, a diminished quantity of sooty-looking urine and the discovery of red and white blood-corpuscles will render the diagnosis positive. The history of the case and the causal factors are also to be taken into consideration.

**Prognosis.**—The *duration* of ordinary exudative nephritis following exposure to cold and wet varies from a few days to three, four, or six weeks. The albuminuria steadily decreases, and with the casts finally disappears, while the daily quantity of lighter urine increases, as does the daily excretion of urea. The prognosis depends much upon the primary disease or causative condition, and also upon the intensity and character of the renal inflammation. Scarlatinal nephritis is less likely to be recovered from than nephritis due to exposure to cold after alcoholic excesses. The acute parenchymatous degeneration that accompanies typhoid fever, diphtheria, and other infectious fevers, as well as pregnancy, is usually a mild affection and recovery takes place easily. But in acute yellow atrophy, yellow fever, cholera, and in severe phosphorus- or mercurial poisoning death may occur from the intense and widespread necrosis of renal epithelium. In favorable cases of ordinary exudative nephritis the dropsy and albuminuria gradually diminish, while the color of the skin and the quantity of urine and urea increase, so that in the course of from three to four or six weeks recovery is established. After the disappearance of the dropsy the albumin may persist for some time, and then slowly disappear; but rarely, in unfavorable cases, even when dropsy has disappeared, albuminuria may continue and the affection become a chronic parenchymatous nephritis.

Serious and often dangerous symptoms of acute nephritis are—severe general edema, dropsical effusions into the serous sacs (as hydrothorax), uremia (especially when beginning with cerebral manifestations, as coma or convulsions), and finally inflammation of the internal organs, as pleuritis, pneumonitis, pericarditis, peritonitis, and meningitis. In the absence of uremia recovery in cases of marked general dropsy is quite common. Suppression of urine, however, lasting more than twenty-four or forty-eight hours, is usually a fatal symptom. The prognosis is unfavorable also in cases in which the nephritis has a productive character. Life may, on the other hand, be prolonged for several years.

**Treatment.**—I shall not include here the management of the primary affection of which the nephritis may be either a complication or consequence.

Since the renal function is diminished by the congestion and inflammation, the first object in the treatment is to relieve these conditions and thus restore the excretory function. The single or combined use of diaphoretics and cathartics is practised, therefore, not that the skin and bowels should be made to perform the work normally done by the kidneys, but in order to restore the functional equilibrium by the antiphlogistic effect produced.

Absolute rest in a warm bed and in a warm room is of primary importance. Woollen underwear and blankets should be provided, so as to promote a constant free action of the sweat-glands. These hygienic



measures should be carried out both in the mild and in the severer cases.

Bland liquid foods only should be allowed in the diet, and the patient should be encouraged to drink freely of water (plain, distilled, or carbonated), lemonade, skimmed milk, or buttermilk; these are especially valuable when hot. Later, thin meat-broths may be allowed, although a strict milk diet is better.

Local bloodletting, as by leeches or cupping over the loins, I seldom employ; in rare cases, however, when much pain is complained of, it may be useful, although hot fomentations may be more so. Diminution of the edema and the elimination of urea and other urinary constituents that may be retained in acute nephritis are best obtained by exciting a profuse perspiration. The hot-air or hot-water bath and the hot wet-pack may be used to accomplish these results, and in most cases the last-named method suffices. It is easily applied by wringing a blanket out of hot water, wrapping the patient in it, and then with a dry blanket, and finally a rubber-cloth cover, surrounding all. This furnishes a steam-bath in which the patient may remain until copious sweating has lasted an hour or so, according to the condition. Children suffering from scarlatinal nephritis may be treated thus, or quite readily also by immersion in hot water, for twenty, thirty, or forty minutes; the skin should then be lightly dried, and the child wrapped in warm sheets or blankets and warmly covered in bed. Hot vapor or air may be generated alongside the bed, and transferred under the raised or cradled bed-clothes by means of a tin funnel and pipe. The sweating will be aided by the drinking of hot lemonade or soda-water or of water containing spirit of Mindererus. Should the skin fail to respond to these measures, as in uremia, perspiration may be started by a hypodermic injection of pilocarpin (gr.  $\frac{1}{8}$  to  $\frac{1}{6}$ —0.008 to 0.0108), after which it will continue to pour out on the application of heat. The heart and pulse should be watched after the injection of pilocarpin, as serious collapse sometimes attends its use. The sweatings should be repeated until the drowsy disappears and as often as the patient's strength will permit. A useful adjunct to the above is the administration of hydragogues, as the saline cathartics, elaterium, and compound jalap powder. Elaterium extract (gr.  $\frac{1}{6}$ — $\frac{1}{4}$ —0.0108–0.0162) is prompt in action, and magnesium or sodium sulphate (ʒj—4.0), given in hot concentrated solution every hour, or a calomel purge, may be recommended. It may be necessary to aid in relieving the tension and distress of extreme edema by multiple punctures or by the use of a small trocar and canula, with a drainage-tube (Southey) attached to the latter after the trocar is withdrawn. Aspiration must be performed if either hydro-thorax, hydro-pericardium, or ascites assumes serious proportions. Half-ounce (16.0) doses of the spirit of Mindererus (liq. ammon. acetat.) in water may be added to the diaphoretic treatment; this, combined with aconite, aids in controlling the fever that may be present and in preventing the vaso-constriction that is often premonitory of uremic symptoms.

Uremic convulsions that do not soon yield to prompt diaphoresis and catharsis should be treated by venesection. As much as a pint or two (.5–1 liter) of blood may be withdrawn and life saved thereby. Sometimes chloroform-inhalations are needed to subdue the very violent con-

vulsive seizures, as in eclampsia. Their return may be prevented by rectal injections of potassium bromid and chloral, consisting of 1 dram (4.0) of the former and  $\frac{1}{2}$  dram (2.0) of the latter.

Nausea and vomiting may be controlled by the use of cracked ice, minute doses of cocain, dilute hydrocyanic or hydrochloric acid, bismuth, and by the addition of soda- or lime-water to the milk.

Contraction of the arteries with increased tension and beginning muscular twitchings call for the use of nitroglycerin, chloral hydrate, or, possibly, morphin.

Diuretics other than the simple diluent drinks mentioned have very little use in the therapy of acute diffuse nephritis, at least early in the disease. Later, as adjuvants to the diuretic properties of water, potassium bitartrate or acetate, sodium benzoate, and cardiac stimulants, as caffein citrate and the infusion of digitalis, may be given, well diluted.

During convalescence care must be exercised that the patient does not catch cold. The diet must not be increased to solids too suddenly nor too rapidly, and particularly in the matter of meats. Light watery vegetables, fruits, and cereals may be gradually added to the diet-list, although milk should be mainly used. Ferruginous tonics are indicated for the anemia, and Basham's mixture is an admirable preparation at this stage.

A change of locality to a warmer, drier, and more equable climate, and careful habits of dress, diet, and exercise, are necessary in cases of recovery from the very serious forms of nephritis, in which the renal parenchyma is shown to have been somewhat damaged by the persistence of slight albuminuria at intervals.

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## CHRONIC NEPHRITIS (EXUDATIVE).

(*Chronic Bright's Disease; Chronic Parenchymatous Nephritis; Chronic Diffuse Nephritis with Exudation; Chronic Tubal and Chronic Desquamative Nephritis; Chronic Glomerulo-nephritis; Large White Kidney; Secondary or Fatty and Contracted Kidney.*)

**Definition.**—A chronic diffuse inflammation of the kidneys, attended with epithelial degeneration, exudation from the blood-vessels, and permanent connective-tissue changes in the stroma. According to Delafield, this is the *chronic productive (or diffuse) nephritis with exudation*—one of two varieties of chronic Bright's disease.

**Pathology.**—Although there are several types of pathologic kidney in this disease, and many individual cases in which anatomic differences are noted, the changes of structure are essentially the same, and the variations depend upon the causation and duration of the nephritis.

The first type of kidney to be mentioned is the *large white kidney* (without waxy degeneration). It is either enlarged or normal in size, and pale or yellowish in color. The surface is smooth, and the capsule is easily stripped off. On section the cortex is broader than normally, yellowish-white throughout, or it may present opaque yellowish or whitish areas with mottlings of red. The pyramids are congested in some

cases. Microscopically, the following changes are commonly observed: the renal epithelium is swollen, hyaline, granular, or fatty, and more or less disintegrated or flattened; the glomeruli are enlarged from the growth of the capsule-cells and of the cells covering the capillaries, and in some cases, owing to the connective-tissue thickening of the capsule, the tuft of capillaries is found to be atrophied. The interstitial tissue shows some thickening of the arterial walls and a moderate growth of connective tissue in patches around the glomeruli and tubules; the latter contain hyaline and granular casts.

The *small white kidney*, or secondary contracted kidney, in most instances is probably a later stage of the preceding, in which the degeneration of epithelium is more advanced and the growth of connective tissue and resultant cicatricial contraction are prominent features. The kidneys are about normal in size (shrinkage of the large white kidney), the surface is slightly granulated, and the capsule is proportionately adherent. While this kidney is usually grayish or yellowish in color (*pale, granular kidney*), there may be some mottling due to red spots. The consistence is firmer than that of the large white kidney. The cut-surface shows yellowish-white foci of the fatty degenerated epithelium in the somewhat narrowed cortex, and hence the term that is sometimes used of "small, granular, fatty kidney." Under the microscope we find extensive degeneration and disintegration of the epithelium of the glomeruli and convoluted tubules, with atrophy of the parenchyma, and a corresponding increase of the interstitial connective tissue. Waxy degeneration may be associated.

Another variety is the *large red or variegated kidney of chronic hemorrhagic nephritis*. The organs are usually enlarged, swollen, red, and congested-looking or mottled, and frequently "bumpy" or slightly bossellated. The capsule is slightly adherent to the depressions between the bosses. Red spots, due to small hemorrhages, may be noticed on both the outer and cut-surfaces of the kidney. The section shows also congested portions and gray or yellow spots corresponding to the anemic and fatty degenerated portions. Small cortical hemorrhagic areas or striations, brownish-red in color, are distinctive of the kidney. The microscopic appearances are those of the large white kidney plus those of acute nephritis. Or, there may be inflammatory edema and cellular infiltration of the intertubular tissue, and dilated tufts of capillaries with surrounding cellular hyperplasia. This variety of chronic nephritis is frequently found in inebriates.

**Etiology.**—The disease may follow either the acute diffuse nephritis, as of scarlet fever or pregnancy, or simple chronic congestion and chronic degeneration of the kidneys. More often it arises insidiously, in a subacute manner and without any previous acute manifestation. Males are more frequently subject to this form of chronic Bright's disease than females. Children affected with the disease have usually had scarlatinal nephritis. Young adults are more commonly affected, however, with the usual variety, developing subacutely. Drinkers of beer and other malt and alcoholic intoxicants seem to be liable to the disease. It is not improbable that some toxic or infectious agency, acting slowly and persistently, may in the insidious cases be the cause of the nephritis, although manifestations elsewhere may be absent. I have observed it



in certain individuals living in malarial regions. Persons working under exposure to cold and wet, or those living in humid and low, marshy localities, are more liable to this renal malady than those who are better protected from climatic vicissitudes. Tuberculosis, syphilis, and chronic suppuration may give rise to this so-called "parenchymatous" form of chronic Bright's disease, and it is usually combined with amyloid disease (waxy degeneration).

**Symptoms.**—There may be a persistence, in a lesser degree, of the symptoms of an acute parenchymatous nephritis, particularly the anemia, dropsy, and the albuminuria, until the affection becomes chronic. In most cases, however, the disease develops slowly and gradually, in a *subacute* manner, though the earlier symptoms seldom indicate any renal derangement. There may be simply a general impairment of health and strength, loss of appetite, nausea, and attacks of indigestion, headache, dulness, and perhaps some pallor. Soon there is puffiness of the eyelids or swelling of the feet or ankles, or both, and the complexion takes on a blanched appearance. The *edema* gradually extends up the legs, and is often worse as the day grows, while on rising in the morning it may be found to have disappeared during the night's rest and recumbency. The *quantity of urine* is diminished in the majority of cases, though in the later stages it may be nearly or quite normal, and even slightly increased in long-standing instances of pale contracted kidney or when absorption of the dropsy is taking place. Superadded acute nephritis may cause a very scanty or a suppressed secretion of urine. The specific gravity is, of course, increased in scanty urine, and *vice versâ*. *Albuminuria* is often quite marked. The amount of albumin may be from one-fourth to three-fourths of the volume of the urine, or from 1 to 3 per cent. by weight, so that the daily loss of albumin may be considerable. The urea is much diminished. The color of the urine is turbid, sometimes smoky-yellow, and urates, casts, red and white blood-cells, epithelial cells, granular debris, and fatty granular cells are found in the usually abundant sediment. The *tube-casts* are of different varieties, but narrow or broad hyaline, fatty granular (Fig. 65), and epithelial casts are commonly observed.

The *edema* is prominent and persistent. It gradually extends all over the body, so that pitting can be obtained on the limbs, chest, abdomen, and back. The loose subcutaneous tissues, as of the penis, scrotum, and eyelids, are particularly distended. In chronic hemorrhagic nephritis, only, the edema may be absent or very slight. The *pasty, pallid complexion* and *anasarca* are most characteristic of chronic exudative nephritis, especially with large white kidney. The dropsy may be moderate and about stationary for several months; then, despite all treatment, it becomes insidiously worse, death ensuing in a month or two.

*Dropsy of the serous sacs*, with its attendant distressing symptoms, may be present in serious cases, and edema of the larynx and lungs may come on suddenly and cause death. Dyspnea may be toxic and nervous, as well as mechanical or cardiac in origin. *Cardiac dyspnea*, due to failure of the heart's action, as seen in many cases, is usually worse on lying down. It may be provoked by vaso-constriction, and is then a danger-signal of uremia. Catarrhal bronchitis may be associated with cough and expectoration.

The heart is often affected with *moderate hypertrophy of the left ventricle*, and later by dilatation and weakness of both ventricles. The *aortic second sound is accentuated* and the *pulse-tension increased*.

*Uremic symptoms* are frequently manifested, except the convulsions which are common to chronic nephritis without exudation. Headache, vertigo, sleeplessness, nausea and vomiting, diarrhea, and stupor, coma, or delirium, may develop and precede a fatal termination.

*Albuminuric neuro-retinitis*, as evidenced by dimness of vision and field-defects, occurs in quite a number of cases. The skin of the legs becomes subject to a red eczematous eruption in some cases of great dropsical distention. In the absence of complicating inflammations, such as pericarditis, endocarditis, pneumonitis, and ulcerative colitis, which are rare, the temperature is practically normal.

The **course** of chronic exudative nephritis may either continue from bad to worse, until death ends all in a year or two, or anemia, dropsy, and albuminuria may attack one who for years previous has had apparent good health, after a first attack the second proving fatal within a few months. Again, some patients, having a little pallor, slightly diminished urine of high specific gravity, with albumin, may complain of nothing for years, until decided attacks, lasting for several months, may occur, with intervals during which the dropsy, dyspnea, etc. may be absent, although some albuminuria persists.

The average *duration* of the disease varies from one and a half to three years. The duration of chronic hemorrhagic nephritis may be somewhat longer (eight months to two years) than that of the large white kidney (six to eighteen months), but it is shorter than the secondary, contracted kidney, which lasts from one and a half to three or even five years.

**Diagnosis.**—The diagnosis of the disease itself is not difficult, but of the stage or the variety of kidney it is almost impossible to tell correctly in some instances. The urinary examination, coupled with the symptoms of dropsy and anemia, is sufficiently diagnostic of chronic diffuse nephritis (with exudation).

In cases of *large white kidney* the urine passed is less in quantity and of higher specific gravity than in the small, pale, and contracted kidney. Edema is usually greater in the former also, while in the latter cardio-vascular changes are more marked, as shown by the physical signs and the hard pulse. The transition of the disease from the earlier to the later stage may be thus noted. The casts in the latter may also be narrower and more darkly granular than in the large white kidney. The existence of *hemorrhagic kidney* may be inferred from the history of alcoholism and the persistent presence of red blood-corpuscles and blood-casts in the urine.

Chronic parenchymatous is distinguished from chronic interstitial nephritis by the following points of difference:

#### CHRONIC PARENCHYMATOUS NEPHRITIS.

Occurs in early or middle life.

There is a previous history of an acute attack of scarlet fever, or perhaps of acute alcoholism.

#### CHRONIC INTERSTITIAL NEPHRITIS.

Occurs later in life.

A previous history of gout, chronic lead-poisoning, syphilis, excessive eating and drinking (spirits), nerve-strain; otherwise often negative.

## CHRONIC PARENCHYMATOUS NEPHRITIS.

The onset is gradual or markedly manifest.

Dropsy is a constant symptom.

Vascular changes and cerebral symptoms are comparatively uncommon.

Marked albuminuria, with tube-casts.

Urine but little increased in quantity, often diminished; specific gravity is increased or slightly diminished.

Anemia occurs earlier and is more distinct.

Uremic symptoms are generally less severe—amaurosis, vomiting, diarrhea, headache.

Runs a shorter course—from two to six or seven years.

## CHRONIC INTERSTITIAL NEPHRITIS.

The onset is very slow, insidious, and indefinite.

Dropsy is rare.

Arterio-sclerosis, cardiac hypertrophy, and cerebral symptoms are common.

Very slight albuminuria and few casts.

Urine of very low specific gravity, and excessive in quantity.

Anemia slowly progressive and less marked.

Uremic symptoms are, generally severe—coma and convulsions, great dyspnea.

Has a more chronic course—seven to thirty years.

**Prognosis.**—This is invariably bad as to cure, though life may be prolonged in certain cases. In severe cases death may take place in from three months to a year, either from uremia, dropsy, cardiac dilatation, or complications. Cases of a year's duration almost never recover, and, *a fortiori*, those in which advanced secondary contraction of the kidney may be inferred are incurable, and may soon terminate fatally. Complete recoveries from the disease, particularly in children that have had scarlet fever, may occur but rarely. The prognosis depends greatly on the quantity of urine passed in the twenty-four hours, the excretion of urea and total solids, and upon the amount and persistency of the albumin, as well as upon the degree of cardio-vascular and retinal changes.

**Treatment.**—The indications for treatment are similar to those in acute nephritis. The dropsy and uremia must be treated symptomatically, and the *diet* is of importance. Skimmed milk and buttermilk, with dried bread, crackers, and zwieback, perhaps, should be depended on as much as possible when dropsy is pronounced. When dropsy is slight, more solid food, white meat, vegetables, rice, and other light cereals, and fruits, and an out-of-door life should be recommended.

Residence in a warm, dry climate may aid in prolonging life. Woolens should be worn next to the skin, and prolonged, sudden, and severe exercise should be forbidden.

The infusion of digitalis, strophanthus, spartein, or convallaria, may be needed in cardiac weakness, or nitroglycerin for contracted and tense arteries with a tendency to uremic twitchings. Unirritating diuretics and Basham's mixture for the anemia are useful. Strontium lactate in doses of from 15 to 20 grains (0.972–1.29), three times daily, I have found useful in some cases. Diuretin has lately been tried, also, with favorable results.



## CHRONIC NEPHRITIS (NON-EXUDATIVE).

(*Chronic Interstitial Nephritis; Chronic Bright's Disease; Primary, or Genuine, Contracted Kidney; Cirrhotic Kidney; Red Granular Kidney; Renal Arteriosclerosis; Chronic Productive (Diffuse) Nephritis without Exudation (Delafield); Gouty Kidney.*)

**Definition.**—A chronic diffuse inflammation of the kidneys, attended with a growth of connective tissue in the stroma, degeneration and atrophy of the renal parenchyma, and marked change in the cardiovascular system.

**Pathology.**—In genuine primary contraction of the kidneys there is a reduction in size and weight about equal in both organs. They may be only one-half or one-third the size of normal kidneys, and the two kidneys together may not weigh over two ounces. They are often found imbedded in thick adipose tissue, the capsule being thick, opaque, and very adherent, so that on stripping it off it brings away portions of the renal cortex. The outer surface of the kidney is red, irregularly granular, or finely nodular, and occasional small cysts are sometimes present. The consistence is firm, dense, and resistant to the knife. Examination of the cut-surface shows a thin atrophied cortex, and dark, reddish streaks alternating with pale portions. The pyramids are also diminished, and darker than the cortex. In the gouty contracted kidney the pyramids show fine striations of sodium urate or of uric acid, or crystals representing uric-acid infarctions.

*Microscopically*, the essential changes are an increased production of connective tissue, especially in the cortical substance, and a more or less proportionate degeneration and atrophy of the renal parenchyma, the destruction of which is due to the circulation of noxious agents, but which is replaced by cicatricial fibrous tissue (Weigert).

The new tissue is not uniformly distributed in the cortex, but occurs in irregular masses around the shrunken glomeruli or between the tubules. The distribution of connective tissue in the pyramids is more diffuse. Many of the glomeruli are quite small and fibrous in advanced cases, while in the earlier cases the cells of the tufts and capsules are swollen and multiplied, and a small-celled infiltration is seen around the glomeruli and tubules. Later this infiltration of cells becomes fibrillated and ends in thickening. Glomerular atrophy is due partly to the changes in and growth of the capillary and intra-capillary cells, as well as of those around the tufts; partly also to capsular thickening and hyaline or waxy degeneration; and partly to the thickening and occlusion of arterioles.

The tubules show marked changes. Some are included in masses of connective tissue, so that there is compression-atrophy and even total obliteration of the lumen. In other instances the intertubular connective tissue constricts the tubules in certain places, so that the lumen is elsewhere increased. This dilatation is especially prominent in the granules seen on the outer surface of the kidney, and, owing to the damming back of urine in some of the tubules thus obstructed, little cysts are visible to the naked eye here and there. The epithelium lining these tubules shows granular, fatty, or waxy degeneration, and may be either flattened,

cuboid, or swollen. The tubes may contain granular or fatty débris and tube-casts.

An important change in most cases is the growth of fibrous tissue in the walls of the arteries, causing sclerosis. This affects the intima (endarteritis), the media, and adventitia, all of which are thickened by the hyperplasia of connective-tissue elements. The arteries and capillaries are thus mostly occluded by the obliterating endarteritis or by their conversion into masses of connective tissue. Waxy or hyaline degeneration is seen also (*vide* Arterio-sclerosis). These arterio-capillary changes may in some cases be the primary condition that leads to granular and contracted kidneys, and may represent the renal effects of a general arterio-sclerosis or fibrosis.

An almost constant accompaniment of chronic, non-exudative, productive nephritis is cardiac hypertrophy. The degree of the latter depends upon the extent of the renal, and also of the general arterial, degeneration and sclerosis. The whole heart may become so large that the term *cor bovinum* has been fittingly applied to it. In moderate enlargements the left ventricle only is hypertrophied.

Complicating lesions of chronic Bright's disease that may be mentioned are cerebral hemorrhage, cirrhosis of the liver, pulmonary emphysema, chronic endocarditis, chronic endarteritis, pericarditis, bronchitis, and gastric catarrh.

**Etiology.**—The cause of the very slow primary, diffuse degeneration, atrophy, and fibroid contraction of the kidneys is sometimes quite obscure. (a) In some cases it would seem to be “only an anticipation of the gradual changes which take place in the organ in extreme old age” (Osler)—the “senile kidney.” (b) *Heredity* undoubtedly plays a part in the causation of certain cases, even to the third or fourth generation. (c) *Age and Sex*.—The disease is more common in males than in females, and it usually begins near middle life; it is seldom manifested symptomatically until about fifty or sixty years of age, and is therefore an affection of advanced life. (d) Individuals having a special tendency to sclerotic degeneration of the arteries, from whatever injurious influence, are more liable to chronic interstitial nephritis, although the prolonged irritation of deleterious (especially chemico-toxic) agents may give rise to the disease in those whose cellular nutrition is usually not defective. Thus, the disease has been attributed to the following causes: alcoholism, uric acid, and lead, giving rise to chronic poisoning. Chronic syphilis and chronic malaria probably are also causative factors. (e) Habitual overeating and drinking, owing to the imperfect metabolism of the substances ingested, cause a constant excretion of irritating products by the kidney, and no doubt frequently cause granular atrophy and sclerosis of the organ. The continuous and even moderate use of alcohol for many years, especially of spirituous liquors, is a widespread cause of the disease. It is equally likely that the excessive use of red meats in the diet leads to the production of the uric acid that induces the renal disorder (*uricemia*; *lithemia*), by deranging the function of the liver (Murchison). (f) Allied to the above is gout, which causes chronic Bright's disease—in England perhaps more than in this country, lithemia and nervous dyspepsia being more common there. (g) According to Strümpell, severe acute articular rheumatism is some-

times followed by contracted kidney. (*h*) Chronic Bright's disease with renal sclerosis is favored in origin and development by the anxieties, worries, and high nervous tension connected with modern business activity and "social functions," the latter particularly acting their part among elderly ladies. Associated with these are usually over-indulgence in rich foods and wines, and sedentary habits. (*i*) The cold, moist climate of New England and the Middle States would seem, according to Purdy, to predispose to contracted kidney. A chronic productive nephritis without exudation, though not the true "contracted and red granular" kidney, may be caused by hydronephrosis, chronic pyelitis, and chronic congestion of the kidney, as from heart-disease.

**Symptoms.**—These may be latent for years, while the morbid productive changes in the kidneys are slowly effected. The first symptoms may not appear until late in life, although the kidneys may be in an advanced stage of degeneration. Or some complicating or intercurrent affection may set in, as pneumonia or pericarditis, and cause the development of grave or fatal renal symptoms. More commonly, however, there is an attack of *uremia*, with headache, stupor, or convulsions, dyspnea, nausea and vomiting, and a tense pulse. This attack may be recovered from. Then there is an interval of variable duration, during which the health is more or less impaired, and lassitude, drowsiness, disordered digestion, headache, failing vision, dyspnea, and frequent micturition are complained of. This is followed by another uremic attack, severer than the first, or perhaps fatal; if not fatal, the general health is still more reduced, and confinement to the house or bed is necessary, until the vital forces can no longer compensate for the destruction of the renal parenchyma.

*Spasmodic dyspnea* (uremic; cardiac) is sometimes the first manifestation of contracted kidney. The gradual onset of periods of uncontrollable drowsiness during the day is often marked. An attack of hemiplegia may also be the first indication of renal disease. Sometimes progressive loss of flesh and strength, with a *dry, harsh, wrinkled skin*, may be from the beginning the only clinical features of the affection until death results from sheer feebleness and emaciation. The complexity and variability of the symptoms make it best to describe them under the heads of the various systems:

**Urinary System.**—The daily *quantity of urine* is usually increased so much that patients are troubled with a desire to urinate frequently, not only during the day, but two or three times during the night. This complaint may be aggravated by the hyperacidity of the urine and the irritability of the prostate (especially in advanced age) that are so often associated with cases of renal cirrhosis. The urine voided during the twenty-four hours may measure several quarts (2 to 4 liters) in well-marked cases of the disease. Early in the attack, when the incipient degeneration and destruction of the parenchymatous cells is taking place, the quantity of urine may be slightly decreased; but as the "blood-flow to the parts that remain must, *cæteris paribus*, be as great as it would have been to the whole of the organs if they had been intact," excessive pressure is brought to bear within the capillaries by the compensating hypertrophy of the heart, and the secretion of the urine, especially of the watery elements, becomes more active. The *polyuria*



may give rise to a suspicion of diabetes. The urine is clear and pale-yellow in color, the *specific gravity* being seldom above 1010 or 1012, and it may be as low as 1002 or 1005. *Albumin* is found only in traces or it may be absent altogether (*glomerular atrophy*), especially in urine voided in the early morning. The urea is diminished, as in all forms of Bright's disease, and there is little or no sediment. A very careful microscopic examination may reveal a *few*, usually *narrow*, *hyaline* or *granular casts*, perhaps some *leukocytes*, and rarely a few *erythrocytes*. In the later stages of the disease or upon the supervention of an uremic exacerbation or of a complicating inflammation the urine may be decreased, the albumin increased, and numerous casts be discovered in a more apparent urinary sediment. Hematuria is rare.

**Circulatory System.**—The *physical signs* of cardiac hypertrophy are present. Symptoms referable to the heart are absent, unless dilatation and feebleness, sudden arterial contraction, cardiac complications, or endocarditis occur. *Inspection* and *palpation* of the *hypertrophied heart* show an apex-beat displaced downward and to the left, and an increased, heaving, and rather circumscribed apical impulse. These signs may be less evident both in cases of coexisting emphysema and later when dilatation may eclipse the hypertrophy. The left border of the deep cardiac dulness extends outside the nipple-line in the fifth or sixth interspace. The first sound of the heart is loud and may be duplicated. A distinctive auscultatory sign is the *accentuation of the aortic second sound*, indicating increased vascular tension; it may have a metallic quality. In quite a majority of the cases I observe, sooner or later, a mitral systolic murmur; it is due to relative insufficiency.

The *pulse* is *increased in tension*, and is hard, incompressible, and persistent, the duration of each pulse-wave being increased (*pulsus tardus*). The radial artery itself—and this is true of most of the palpable arteries—feels hard, thickened, and often tortuous, on account of the arterio-sclerosis. As soon as compensation of the heart fails, symptoms of breathlessness (especially on exertion), palpitation, and the like, appear, and sometimes in paroxysmal attacks ("cardiac asthma"). The resultant stasis gives rise to transudation into the lungs (bronchorrhea; pulmonary edema), and later to edema of the extremities.

**Respiratory System.**—Epistaxis may be a serious symptom. Sudden edema of the larynx may also occur, and is always grave. Transudations into the pleural sac (hydrothorax), as well as into the lungs (*vide supra*), may precede death. Dyspnea, which is either cardiac or uremic, is usually worse at night, and a true orthopnea, together with Cheyne-Stokes breathing, may be observed toward the end of the patient's life and in association with uremic stupor and coma.

**Nervous System.**—Symptoms referable to the nervous system are very important, since they are usually indicative of grave uremia. Cephalalgia is frequent, and neuralgic pains throughout the body, and insomnia, may be complained of. Later great *drowsiness* is often a premonition of uremic coma. Convulsions may be preceded by muscular twitchings, which should attract attention to the imminent danger of the former. Cerebral apoplexy with hemiplegia may be the first symptom of contracted kidney. It is especially apt to occur in cases of marked hardening and weakening of the arteries. There may be

an hemorrhagic pachymeningitis, as well as a hemorrhage into the brain-substance. The hemiplegia may persist until death; or it may disappear in a short time, and be followed by subsequent attacks at intervals. *Formication*, *numbness*, and pallor of one or more fingers (the so-called "dead finger") I believe with Dieulafoy to be sometimes the earliest symptoms of chronic Bright's disease.

Of the **special senses**, *nephritic retinitis* is often the earliest evidence of chronic Bright's disease. The patient may or may not have had slight dimness of vision (mistiness) prior to the ophthalmoscopic examination. The loss of vision affects both eyes, and is usually partial (*amblyopia*). Sudden and complete blindness may come on in grave cases—*uremic amaurosis*—the condition being due to neuro-retinitis. The optic papilla is swollen, and surrounded by retinal hemorrhages or by white dots and streaks ("feather-splashes"). Tinnitus aurium, deafness, and *vertigo* are not uncommon.

**Digestive System.**—Anorexia, nausea, and annoying dyspepsia are often complained of. Severe vomiting may usher in an uremic attack. Catarrhal gastritis may exist for some time, the tongue being coated and the breath heavy and urinous. *Uremic diarrhea* may also occur.

**The Skin.**—Edema is usually absent in renal sclerosis; when it does occur, however (as in the ankles and limbs), it is due to dilatation and failure of the heart. The skin is dry, and minute lustrous scales of urea may be seen around some of the pores. A certain degree of pallor is noticed, and often the skin has a cyanotic tinge. *Pruritus* and troublesome eczema are frequently present, and *muscular cramps*, occurring especially in the calves of the legs and at night, may also be associated. The general nutrition gradually fails, so that in advanced cases the debility and emaciation are extreme.

It is important to bear in mind the fact that *uremia* may come on at any time during the course of the disease, and that it may be the first symptomatic manifestation; also that it may either be sudden and severe in its onset (acute uremia) or mild, insidious, and gradual (chronic uremia). Moderate fever may attend an uremic attack, or the temperature may be normal; in chronic uremia, with prostration, coma, delirium, and feeble pulse, it may be even subnormal.

Among the **complications** that may occur in the red, granular, and contracted kidney are the following: pneumonia, either lobar or lobular; pleuritis, pericarditis, laryngitis, bronchitis, gastritis, enteritis, peritonitis, meningitis, endocarditis, emphysema, phthisis, and hepatic cirrhosis.

**Diagnosis.**—This depends in great part upon the physical, chemical, and histologic examination of the urine. Both the morning and evening urine should be examined repeatedly for albumin and casts, since one examination—and especially that of the morning urine—may give negative results, owing both to the scarcity of these two pathologic elements and to the fact that albumin may be altogether absent in some instances. The mere discovery of a trace of albumin or of a few casts is not always positive evidence of chronic Bright's disease, as both may exist in other conditions. But the age, habits, and symptoms of the patient must be studied in connection with frequent urinary examinations; and a persistent slight albuminuria, with casts, and the

passage daily of large quantities of clear, pale urine of low specific gravity, afford sufficient grounds for making the diagnosis.

Contracted kidney should be suspected in all cases in which, during middle life, either one or more of the following symptoms and signs may be noticed: frequent headache, congestive disorders, repeated epistaxis, vertigo, dimness of vision, intractable conjunctival irritation (Allerman), impaired strength, dyspneic attacks, gastro-intestinal dyspepsia, noises in the ear, itching of the skin, cramps in the calves, muscular twitchings, growing mental dulness, increasing pulse-tension, and rigidity and tortuosity of the temporal and radial arteries. Sudden coma, convulsions, amaurosis, apoplexy, vomiting, or dyspnea in persons in the middle period of life, with or without a history of polyuria, should create the suspicion of chronic Bright's disease. It will be found in such cases that there has been a diminution in the urinary flow before the attack. Persons of lithemic, gouty, rheumatic, or alcoholic habits, or in whom lead-toxemia is discoverable, with evidences of cardiac hypertrophy, an accentuated aortic second sound, and a hard pulse, are often readily diagnosed as subjects of contracted kidney when a further examination of the urine is made.

The diagnosis may be very difficult, however, in cases in which the first examination of the patient is made during a sudden uremic or apoplectic attack. Catheterization should be done if necessary, and the detection of albuminuria will then clear the diagnosis.

In order to differentiate between primary renal affection with secondary cardiac hypertrophy and *primary heart-disease with a secondary congested kidney occurring late in the case*, the general features, course, symptoms, and signs must be carefully and judiciously balanced. Prominent cardio-vascular changes would indicate an arterio-sclerotic kidney, rather than the primary granular and contracted kidney of toxic origin, though even here the diagnosis is often quite difficult. The symptoms of ordinary non-inflammatory *senile kidney* may not be unlike those of chronic interstitial nephritis, though not so severe; and yet, from excessive eating and drinking at times, uremic attacks may supervene to cloud the diagnosis.

**Prognosis.**—The *duration* of chronic interstitial nephritis varies. In uncomplicated cases it may last for five, ten, twenty, or possibly thirty years. Complications or intercurrent affections may, however, shorten the duration very much, or the existence of the condition may be unknown, as frequently happens, when the *postmortem* examination shows the characteristic kidneys in one who during life had no symptoms indicating renal disease, and whose death was caused by some intercurrent disease. The gradual destruction of the renal parenchyma and its replacement by scar-tissue cause irreparable damage to the organs. On the other hand, the fact that the process is usually a slow one and its duration long is compatible with the preservation of life for many years, and with comparative comfort, even, in many instances. The prognosis in a given case depends very much upon the general constitutional condition, the cardio-vascular state, and the presence or absence of uremia and inflammatory complications. Cardiac dilatation and insufficiency indicate a not far distant end. Convulsive and apoplectic seizures are often fatal, and hemorrhages, persistent vomiting, and diarrhea, *retinitis nephritica*,



coma, and delirium render the prognosis as to further systemic tolerance of the degenerated kidneys exceedingly grave.

**Treatment.**—An early recognition of the disease and the steadfast practice of careful hygienic measures will prevent, to a considerable degree, the advance of the cirrhotic changes. Noxious substances entering into the etiology of the affection must be avoided and removed as far as possible. The formation of uric acid must be reduced by dietetic management, alcoholics must be interdicted, and lead—when the cause of the condition—must be kept from further poisoning the system by a change of occupation. By diminishing these irritants the heart and blood-vessels are also conserved—a point of vital importance.

The **hygienic treatment** must embrace a regulation of all the habits of body and modes of life. The patient must be treated, and not his malady, since that is incurable. A dietary that is suitable for each individual case must be made out, and on general principles. Saundby's rule is a good guide: "Eat very sparingly of butcher's meat; avoid malt liquors, spirits, and strong wines." An exclusive milk diet may be necessary for short periods when gastric irritation is present, but in such a chronic disease undue weakness would result from a restriction to milk alone. I would therefore recommend a light nourishing diet, including lean meat once daily in favorable cases. Vegetables, greens, fruits, and light, well-cooked farinaceous articles may also be partaken of, and tea, coffee, and cocoa may be drunk. The use of natural mineral waters aids in the renal circulation and keeps the kidneys flushed. In general a mixed diet will be of advantage; the nitrogenous and carbohydrate elements (sugars and starches) are used in limited amounts, while pure fats and fruits (raw or cooked) are to be recommended. Stout persons and those leading sedentary lives should have less food than those taking exercise, and gastric disorder requires the elimination of all but soft, bland foods, or a liquid diet until digestion is restored. Extremes of bodily, mental, and emotional activity should be avoided, and physical exercise should be moderate, regular, and taken in the open air, provided the latter be warm and dry. Mental labor should never be excessive, nor should the patient be subjected to the vicissitudes of worry, anxiety, or competitive tension. Venereal excitement and indulgence of any kind tending to unbalance the self-control or disturb the equanimity, cheerfulness, and contentment should be strictly forbidden and guarded against.

A change of residence to a warm, mild, and dry climate is often of service in prolonging life. The variability and humidity of temperate climates, particularly during the winter months, aggravate this disease, while a sea-voyage or a sojourn at some southern European resort may be very beneficial to one who can afford it.

The indications for **medicinal treatment** are principally as follows: The bowels should be kept free by the aid of laxatives or laxative alkaline mineral waters. Papoid, peptenzyme, and other digestants, with bitter tonics, are useful in some cases in which a furred tongue and indigestion are troublesome. Acids or alkalis, according to special indications, may also be used simultaneously. An increased vascular tension (vaso-constriction), such as to place a serious strain upon the heart; the other extreme, of a very low tension that induces dropsy; and compli-

cations, usually uremic (convulsions, dyspnea, headache), also call for therapeutic assistance. High tension is to be met by the cautious use of nitroglycerin in gradually ascending doses, beginning with 1 minim (0.066) three or four times daily, until all danger of rupture of the vessels seems to be past. Headache, vertigo, and the so-called renal asthma (dyspnea) are also often relieved by this drug.

Low tension, with signs of cardiac dilatation, scanty albuminous urine, and edema, requires heart-tonics and stimulants, in conjunction with purgatives. Digitalis (preferably in infusion) has good effects, especially when combined with strychnin nitrate or with caffein citrate. Calomel and the salines should be given for the dropsy.

Uremic symptoms should be treated as in acute Bright's disease by causing profuse sweating and free catharsis, and in some cases by phlebotomy. Inhalation of amyl nitrite or chloroform, or, what is often a useful and necessary measure, the hypodermic injection of morphin (gr.  $\frac{1}{6}$ –0.0108), may be tried in convulsions, severe headache, or dyspnea.

Contracted kidney of a probable malarial or syphilitic origin may be benefited somewhat by the use of arsenic and the iodids respectively; but no drugs can possibly restore the destroyed renal parenchyma or transform connective-tissue cells into secreting kidney-cells.

## PYELITIS.

(*Pyelonephritis*; *Pyonephrosis*.)

**Definition.**—Inflammation of the pelvis of the kidney. The compound terms above (in italics) represent an inflammation of the kidney-structure as a result of, and combined with, pyelitis.

**Pathology.**—In the mildest varieties of pyelitis (the catarrhal) the morbid changes consist simply of a reddened, swollen, and turbid mucous membrane, covered with an exudation of viscid muco-pus and desquamated epithelium. Ecchymoses are sometimes seen. The urine in the pelvis of the kidney is also turbid from the admixed pus-corpuscles and pelvic epithelium. In calculous pyelitis, owing to prolonged and severe irritation, purulent inflammation and ulceration prevail, and the kidney-structure is also involved by extension (*pyelonephritis*). Renal abscesses are thus formed, and small dark calculi are frequently found mingled with the pus in quite a number of small abscess-cavities; or perhaps, as noted before (*vide* Nephrolithiasis), one large abscess-cavity may replace the destroyed renal parenchyma (*pyonephrosis*).

A diphtheritic inflammation, with the formation of a false membrane and sloughing of the pelvis, sometimes follows the severe infections of the specific fevers. Marked hemorrhagic areas may be seen also. In tuberculous pyelitis there is usually an association of nephritis with areas of tuberculous softening and ulceration, and later *pyonephrosis*. In very chronic and sluggish cases the pyelitis may be followed by an infiltration of the kidney-structure with cheesy or putty-like masses that may become the seat of calcification.

Persistent obstruction leading to pyelitis is associated with dilatation

of the pelvis from retention of urine or of pus (pyonephrosis). This in turn, from prolonged pressure, causes the marked atrophy of the secreting structure of the kidney that is seen in such cases. There is also an increase in the interstitial tissue and secondary contraction.

The so-called *surgical kidney* is found when an acute bilateral pyelitis, following a severe cystitis, has excited an acute suppurative inflammation of the kidney. Acute suppurative or interstitial inflammation of the kidney due to metastatic or miliary abscesses is considered under the heading *Pyemia* (*vide* p. 199).

**Etiology.**—Pyelitis rarely is primary or independent in origin, as after exposure to cold and wet. The secondary causes of pyelitis are as follows: (1) renal calculi (the most frequent); (2) extension upward of urethritis, cystitis, or ureteritis, particularly when gonorrheal in origin; (3) retention of decomposed urine in the pelvis of the kidney; (4) renal affections, as tubercle, carcinoma, and acute nephritis; (5) specific fevers; (6) foreign bodies, other than stone in the pelvis; (7) irritating diuretics. To point out briefly certain additional facts bearing upon the causation of pyelitis in the order named, it should be mentioned that *calculous pyelitis* may result from the irritation of the constant presence and passage of small stones ("gravel"), or even of uric-acid "sand," as well as from the large dendritic concretions that send offshoots into the calyces. Extensions of inflammation to the pelvis from lower portions of the urinary tract may occur in protracted cases of such affections as gonorrheal urethritis and puerperal and calculous cystitis. *Obstructive pyelitis* sometimes follows the impaction of renal calculi or of other foreign bodies in the ureter when there is pre-existing inflammation of the tract, or when, as usually happens, there is chemical irritation from the decomposition of the accumulated urine. There may be obstruction in the bladder and urethra, as from enlarged prostatic tumors, stricture, phimosis, and paralysis of the sphincter vesicæ, or as in paraplegia. Under the consideration of tuberculosis and carcinoma of the kidney is included the involvement of the pelvis by these conditions. *Infectious pyelitis* may also result from small-pox, diphtheria, typhus and typhoid fevers, and scarlatina, and it depends upon the irritating effect of certain substances eliminated by the kidneys. It is usually associated with more or less nephritis (pyelonephritis). Parasites, such as the echinococcus (hydatids), distoma, strongylus, and filaria, may give rise to pyelitis. Cantharides, cubebs, copaiba, turpentine, and diabetic urine even, may in rare instances also excite a pyelitis.

**Symptoms.**—These are frequently overshadowed by those of the primary condition that causes the pyelitis: they are varied also for the same reason. The clinical manifestations of a simple catarrhal pyelitis are slight pain and tenderness in the region of the affected kidney or kidneys, mild fever, with a *turbid urine of acid reaction*, showing a few pus-cells, a little mucus, rarely some red blood-corpuscles, and a trace of albumin.

In the severer varieties, as in calculous pyelitis, especially when there are attacks of renal colic, the urine frequently shows to the naked eye the presence of *blood* and a marked amount of *pus*, some *mucus*, and the transitional *caudate epithelial cells* from the middle layers



of the mucosa. The presence of the latter, however, is not constant, hence its absence does not exclude the existence of a pyelitis, since some of the most destructive forms of the affection, as the acute or chronic suppurative or the pyelonephritic, may be unaccompanied by the presence of the pelvic epithelium in the urine. This holds still more true in the case of true pyonephrosis, in which the kidney usually becomes one large abscess.

In severe pyelitis the *pain* is often acute, coursing *down the ureters*. The fever is moderate, and there are present the common symptoms described under Nephrolithiasis (*vide* p. 970).

The *fever* in purulent pyelitis (pyonephrosis) and pyelonephritis takes on a *hectic* or *typhoid* type. Paroxysms of rigors or chills, followed by a rapid rise in temperature and ending in perspiration, may be observed; or there may be marked prostration and feebleness of circulation, delirium, and stupor. The temperature-curve runs an irregular course, with marked remissions, in cases having a pyemic nature.

In obstructive pyelitis the urine sometimes flows freely and normally for a while, until the developing pain over the inflamed kidney ends in relief by the expulsion of the obstacle and the passage of purulent urine. This *alternation* of normal with pyoid urine is indicative of a unilateral pyelitis.

*Ammoniacal urine* is met with in *cysto-pyelitis*. *Albuminuria* is decidedly shown according to the degree of pyuria.

In chronic suppurative pyelitis or pyelonephritis the pyuria is variable both in quantity and constancy. *Intermittent pyuria* may be due to the temporary blocking of the ureter by a stone (*vide* Obstructive Pyelitis). The pus is seldom mixed with epithelium in chronic purulent pyelitis. The associated intermittent fever may be like that of tuberculous pyelitis, and marked prostration, anemia, and emaciation are concomitants. Evidences of amyloid change may be revealed in long-standing, chronic cases.

The term *ammoniemia* has been applied to that complexus of nervous symptoms that is supposed to arise from the decomposition and absorption of urinary substances. These symptoms may be similar to the manifestations of diabetic coma.

Distinct *enlargement* and *fluctuation* of the diseased kidney may be determined in some cases of pyonephrosis. This may also be intermittent, being detectable while there is obstruction to the flow of pus, and *vice versâ*. According to A. H. Smith, at the menstrual periods pyelitis may be subject to marked exacerbations, simulating renal colic.

In chronic pyelitis with atrophy of the kidney the onset of uremia may terminate the case. Granular kidney alone may have been simulated by the passage of an increased quantity of urine of proportionately low specific gravity.

**Diagnosis.**—This embraces the discrimination from other affections, and the possible detection of the variety—etiologically considered—of the pyelitis. It is most important to pay attention to the clinical history of any case with a view to the discovery of the cause; also the urinary condition must be carefully studied. In the very nature of this affection it

is often impossible to exclude other affections of the urinary tract, as *nephritis*, *cystitis*, and *urethritis*.

Epithelium from the pelvis of the kidney cannot be distinguished from transitional bladder-cells; but, given the indications of a pyelitis, its calculous cause is at once made clear upon the passage of the characteristic uratic or oxalatic concretions. It may happen that the urine from one kidney is prevented from flowing by the impaction of a stone in the ureter. The urine may now flow clear from the other and vicariously acting kidney until, the stone having given way, it suddenly increases in quantity and changes in character, owing to the return of the morphologic elements of the pyelitis (corpuscles, desquamated epithelium, crystals, and débris).

In women catheterization of the ureters and renal pelves, as described and practised by Pawlik and Kelly, is a most certain method of determining in doubtful cases from which side the purulent urine arises. Palpation of the ureters through the lateral and anterior fornix of the vagina will sometimes reveal thickening and tenderness in cysto-pyelitis, and ureteral distention sometimes may be felt in pyelitis calculosa.

Vierordt mentions having seen in some cases of pyelo-nephritis peculiar hyaline casts "split like a pair of trousers." Casts and albumin are usually present when the kidney-structure is involved by extension of the pyelitis, while marked pain in the region of the kidney indicates predominant pyelitis, though it does not exclude the possibility of coexisting nephritis. Marked vesical irritability points to associated cystitis, but in intense pyelitis with much pus and an acid urine vesical tenesmus may also be troublesome. Tuberculous can be discriminated from calculous pyelitis by finding tubercle bacilli in the pus. The presence of a fluctuating tumor in the lumbar region is significant enough of pus; but it may be difficult to determine whether it is due to pyonephrosis or perinephric abscess, although pyuria and the previous history of pyelitis, as well as the more circumscribed and less edematous character of the swelling of the former, are important distinguishing points.

**Differential Diagnosis.**—The *hemorrhagic pyelitis* of Senator, Delafield, and others, described as occurring in milder forms, and particularly in girls of neurotic types, may be distinguished by the intermittent hematuria and the occasional lumbar pain, lasting but a few days or a week, and followed uniformly by recovery. Digestive disturbances may be prominent in these cases.

Difficulty is sometimes experienced in diagnosing pyelitis when coexistent with cystitis—*pyelo-cystitis*. These affections will not be confounded, however, when it is recollected that their histories differ. There is pain in one lumbar region in the former, and in the bladder in the latter.

According to Rosenfeld, (1) an alkaline reaction is not found in uncomplicated pyelitis; (2) the limit of albumin in the urine, even with severest cystitis, is 0.1 per cent. (maximum, 0.15); (3) if the pus-corpuscles are crenated, and, in the absence of vesical tumor, if the red corpuscles of a microscopic hemorrhage are chemically or morphologically decomposed, pyelitis exists, and especially if non-imbricated, small epithelial cells are practically absent. Stress is laid upon the relation of the albumin-content, which is from two to three times greater with pyelitis than with cystitis.

**Prognosis.**—Renal complications always make the pyelitis a serious affection. Catarrhal cases recover. Calculous pyelitis tends toward chronicity. Pyelo-nephritis and pyonephrosis are apt to end fatally from exhaustion or uremia. Perforation and the discharge of pus into the peritoneal cavity, pleural sac, intestine, and bronchi even, may precede death. The gravity of all cases of pyelitis depends upon the causes and upon the tendency to consecutive suppuration.

**Treatment.**—This varies according to the cause: the latter needs to be removed, its effects counteracted, and its return avoided. The treatment of calculous pyelitis is essentially the treatment of nephrolithiasis. Primary inflammation of the lower portions of the urinary tract must be combated; causes of retention of decomposed urine, as an urethral stricture or enlarged prostate, must be diminished; infectious fevers must be judiciously handled and irritating diuretics withheld.

Local measures are of value in all forms of pyelitis. Hot-water bags, fomentations, poultices, and dry cupping are often of great service. Internally, the use of diluents is to be recommended, especially the alkaline mineral waters, flaxseed tea, barley-water, skimmed and butter-milk, and lemonade.

Potassium citrate, uva ursi, buchu, and pareira brava are sometimes selected for their soothing properties. But, practically, none of the remedies named nor any other drug is of any avail when suppuration is once established. Irrigation by means of Kelly's ureteral catheter may be practised with good results in females. Hypodermoclysis of normal salt-solution may be of sustaining value at critical times in cases of infectious pyelonephritis. In chronic pyelitis salol and the oils of turpentine, sandalwood, juniper, copaiba, and erigeron have been used for their stimulating and alterative effects upon the mucous membrane. Surgical intervention is necessary in severe purulent pyelitis, pyelonephritis, and pyonephrosis.

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## HYDRONEPHROSIS.

**Definition.**—An obstructive accumulation of urinary fluid in the pelvis and calyces of the kidney; it may cause dilatation, pyelitis, or inflammation and atrophy of the renal structure.

**Pathology.**—Hydronephrosis is usually unilateral. The pathologic changes consist of a dilatation of the pelvis of the kidney, associated with a degree of atrophy of the renal tissue depending upon the degree and persistence of the pressure. The accumulated fluid causes flattening and atrophy of the papillæ, and gradually of the tubules and glomeruli, as the dilatation and distention increase, until in extreme cases remnants only of the renal structure remain in the walls of the hydronephrotic cyst. The mucous membrane lining the pelvis and calyces first becomes thinned, and later thickened, by the growth of connective tissue, thus forming the dense sac-wall. There is also a growth of connective tissue in the renal parenchyma, medullary and cortical, a chronic nephritis with degeneration and atrophy of the renal cells being set up.

A *nephrotyc cyst* may be very large, containing as much as several



gallons of liquid. Sometimes in medium-sized sacs the external appearance of the walls may be lobulated; the interior, however, usually shows only partial septa projecting from the wall into the cavity of the sac. The smaller sacs partially enclosed by the membranous septa probably represent the dilated calyces. According to the seat of obstruction one or both ureters may also be dilated. If one kidney is affected, its fellow is often hypertrophied.

The fluid contained in the sac varies in composition, but usually is a clear, thin, yellowish, watery urine. The specific gravity is low, and the reaction is often slightly alkaline. Traces of albumin, urea, uric acid, and salts are found. Turbidity may be present, owing to admixture with pus, blood, or epithelium, but only in instances in which previous inflammatory conditions, as a calculous pyelitis, or subsequent complications of like nature have existed.

**Étiology.**—Hydronephrosis—or, better, *nephrydrosis*—is in most instances secondarily produced by diseases—congenital or acquired—that cause occlusion of the ureter. Probably from 20 to 35 per cent. of cases are congenital (Roberts). In these cases the causal condition is one of stricture, due to obstruction caused by a defective development or malformation in the urinary passage of one or both sides, usually the latter. Thus, there may be a valve-like formation or a very acute insertion of the ureter into the kidney. The dilatation has occasionally become so great in the fetus as to cause considerable mechanical difficulty during labor.

Among adults, women are more often subject to hydronephrosis than men, and especially women who have borne children. The condition may be bilateral, as from a stricture low down and due to gonorrheal urethritis, but more often it is unilateral. The causes of these acquired cases are as follows: (1) Impacted calculi in the ureter or renal pelvis. (2) Disease of the ureteral walls, as inflammatory thickening and cicatricial stenosis from ulcers. (3) Flexion and twisting of the ureter, as from movable kidney. (4) Pressure upon the ureter from without, as by tumors and constricting bands (pelvic adhesions). The gravid and retrodisplaced uterus, uterine and ovarian neoplasms, enlarged and prolapsed spleen, and similar conditions causing compression or traction and obliteration of the lumen of the ureter, are found in this class. (5) Diseases and tumors of the bladder that involve the ureteral orifices, particularly carcinoma, or that cause retention, as prostatic enlargement. (6) Urethral stricture.

**Symptoms.**—These depend somewhat upon the cause and extent of the hydronephrosis. Marked bilateral disease, when congenital, may render the fetus inviable. The unilateral variety may be overlooked for years, and no symptoms may point to the trouble until a tumor can be made out by inspection and palpation, or until the ureter of the remaining kidney may become obstructed and symptoms of uremia supervene. The latter are more apt to come on, and earlier too, in double hydronephrosis.

*Locally*, the patient may complain of frequent and severe *pains* that shoot about the affected loin and downward toward the thigh. Sensations of weight and a dragging discomfort are common. Anorexia, nausea and vomiting, eructations, and irregularity of bowel-action are

associated sometimes. In large hydronephrotic cysts a continuous dull, aching pain only may be felt, or, as is not infrequently the case, the tumor may be absolutely painless. Obstinate constipation may result from compression of the colon, or in moderate enlargements diarrhea may occur from the pressure-irritation.

Usually a swelling is detected in the region of the affected kidney. It gradually increases in size, and in marked enlargements distinct bulging may be visible in the hypochondriac and lumbar regions. *Palpation* reveals a rounded, firm, yet somewhat elastic and sometimes fluctuating tumor. There may be slight tenderness. Dulness on percussion is found over the mass, except where the colon overlies it, when tympany is elicited; this is a characteristic sign of kidney-tumors. Moderate enlargements generally do not descend during inspiration. There may, however, be exceptions to this rule.

The *intermittent* form of hydronephrosis (Landau) is interesting from the variations that occur in the size of the tumors. A *marked diminution is coincident with a more or less sudden increase in the quantity of urine passed*; and, on the other hand, as the tumor gradually enlarges the flow of urine decreases. These cases are in most instances due to movable kidney. *Colicky pains* often usher in the periods of greatest distention preceding the sudden increase in the flow of clear urine. This variety of the affection occurs most frequently in women that have borne children. The *general symptoms* scarcely amount to more than a certain loss of flesh incident to the associated worry and anxiety. The filling of the nephrydrotic cyst, the distention, and the pain and discharge, with subsidence of the tumor, recur with variable frequency. According to Osler: "Among the circumstances liable to cause them are sudden and violent exercise, the jarring and jolting of riding and driving, any fatigue, mental emotions, and errors in diet." The tumor may continue to develop in size for several days after the pain has disappeared. The latter may last from several hours to a day. During the intervals, and after the urine has increased in quantity, gradually or quickly, the patient feels tolerably comfortable, and this sometimes for weeks or months. For obvious reasons the tumor is rather mobile in intermittent hydronephrosis.

The occurrence of chills, fever, and sweats, nausea and vomiting, abdominal distention, and rapid pulse usually indicates suppuration, and pyonephrosis may be the consequence. The urine will then be cloudy and reveal pus, following both discharge and aspiration. A lowered specific gravity and the presence of albumin will be noted when a chronic nephritis has been set up. Increased arterial tension and symptoms of acute febrile or chronic afebrile uremia may be added.

*Hydronephrosis paraplegica* is a form of the disease in which paraplegia develops as a complication.

The *course* of nephrydrosis is usually chronic, with variations and exacerbations depending upon the cause of the affection.

**Diagnosis.**—This is obviously very difficult in cases in which the accumulation of liquid is small. Characteristic signs are the gradual development of a tumor in either flank, as described above, with diminution in the urinary flow, followed by a more or less sudden free discharge and the subsidence of the tumor, with recurrences (as in the in-

termittent variety). When these do not occur and the tumor continuously enlarges, aspiration may be practised to determine whether the mass is solid or liquid; the nature of the latter may also thus be ascertained, whether urinary or not. Ureteral catheterization will determine which is the dry side.

The history of the case and the detection of some causative occlusion will point to the diagnosis.

**Differential Diagnosis.**—The nephrydrotic sac must be distinguished by exclusion from an *ovarian cyst*, *cystic kidney*, and *tumors of the spleen, liver, and gall-bladder*. Very large cysts may be mistaken for *ascites*. Assurance of the presence of the colon over the tumor is diagnostic, and a chemical examination of the fluid obtained by the use of the exploring needle will suffice in most cases. It should be remembered, however, that a slight amount of urea is sometimes found in ovarian cystic fluid. The presence of pus-cells in abundance in the aspirated fluid, with symptoms of suppuration, is significant of pyonephrosis.

**Prognosis.**—This is generally unfavorable, though in unilateral hydronephrosis evidences of compensation on the part of the unaffected kidney should render the case guardedly favorable, particularly if the cause be a movable kidney. The bilateral affection is always grave, owing to the danger of uremia. Infection of the cyst with pus-organisms is usually a fatal complication. Recovery may ensue in rare instances in which a spontaneous discharge of the liquid takes place. Rupture of the sac is unlikely.

**Treatment.**—The removal of the cause is seldom feasible. Symptomatic treatment only is required in mild cases, though sometimes gentle massage over the sac, properly directed and cautiously applied (to avoid rupture), may cause a reduction in the size of the tumor. Most often surgical measures only are of use. These embrace puncture and aspiration, incision (nephrotomy) and drainage, nephrorrhaphy, nephrectomy, and the formation of a renal fistula. These procedures, however, are undertaken only when successive reaccumulations of the fluid follow those measures first mentioned.

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## PERINEPHRIC ABSCESS.

(*Perinephritis*.)

**Definition.**—Suppurative inflammation of the connective tissue surrounding the kidney.

**Pathology.**—The suppuration attacks the lax adipose tissue or the fatty capsule in which the kidney is imbedded and the adjacent retroperitoneal tissue. The starting-point of suppuration is usually behind the kidney. There may be several small abscesses at first, but more often a single large abscess is found. The walls may be soft and shreddy, or in more chronic cases thickened and fibrous. A bulging externally over the affected lumbar region is not infrequent, particularly in large and extensive accumulations of pus. The latter has a tendency



at a given point to burrow into the surrounding tissues, and especially downward toward the iliac fossa, pointing in the groin near Poupart's ligament. It may extend backward and open upon the skin-surface. Sometimes the pus perforates the diaphragm and discharges through the pleural cavity and lungs, or the colon, vagina, bladder, or peritoneum may be perforated. The pus is occasionally quite offensive, and may be ichorous from an admixture of infiltrated urine. Perirenal abscess due to calculous pyonephrosis may contain calculi that have ulcerated through pelvic or renal walls. Thickening of the adjacent peritoneum is often found. In certain cases of perinephritis, which usually gave no symptoms during life, the *postmortem* examination has revealed fibrous adhesions and a firm and thickened and fatty capsule, stripped with difficulty from the true capsule of the kidney.

**Etiology.**—Perirenal abscesses, when not traumatic in origin, develop most frequently as a result of purulent pyelo-nephritis or pyonephrosis. Hence they are *usually secondary*. Other primary conditions that may cause perirenal suppuration are the following: extension of inflammation from the ureter or pelvis of the kidney; from a pelvic abscess; from appendiceal or hepatic abscesses; and from spinal caries (psoas abscess) and empyema. Sometimes tuberculous processes in the kidney and suppurating new growths, as carcinoma and cysts (including the echinococcus), are complicated by perirenal abscess. More rarely such severe infectious diseases as typhus fever, small-pox, and pyemia lead to purulent perinephritis. Finally, there are cases for which no cause is discoverable.

**Symptoms.**—Subjectively, there is noted a *dull, throbbing pain* over the affected region that is increased by motion; sometimes, when the abscess is large and presses on the large nerve-trunks, the pains may become shooting in character and be felt in the leg on the same side. *Numbness* may also be felt. Pain and tenderness on palpation are common. The patient is prostrated, weak, and often quite emaciated, and flexure of the thigh on the affected side is frequent. The characteristic fever of suppuration is present in the deeply remitting or intermitting type, with alternating chills and debilitating sweats. Pus is found in the urine only when the kidney is involved. Sooner or later evidences of a *tumor* are seen; the areas can be palpated, and a gradual bulging in the lumbar area, increasing slowly, with smoothness and glistening of the skin and pitting (edema), may be observed. *Fluctuation* is frequently apparent in advanced cases, and in favorable cases signs of "pointing" appear.

**Diagnosis.**—Should the abscess tend to burrow downward, the condition may be somewhat obscure on account of the absence of distinct local symptoms. Indeed, involvement of the psoas may give rise to symptoms of coxitis, as pain referred to the knee-joint. The diagnosis is usually easy, and when in doubt as to whether the tumor is an abscess or an hydronephrosis or solid mass, the exploring needle should be used.

**Differential Diagnosis.**—An important point in differentiating perinephric abscess from suppurative pyelitis or pyelo-nephritis alone is the fact that in the latter the quantity of urine is usually diminished, whilst in the former there is less apt to be any interference with the

renal secretion. Again, whilst in the latter the urine usually contains blood and pus, in the former the urine is free from blood, though not necessarily from pus, and casts are also absent here.

**Prognosis.**—This is guardedly favorable if the abscess points externally in the lumbar area. Of course rupture into the peritoneal cavity, bladder, bowel, and groin is always a serious occurrence.

The **treatment** is essentially surgical, and consists in free incision and drainage.

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## CYSTIC KIDNEY.

(*Renal Cyst.*)

**Pathology.**—Congenital cystic kidneys are in reality collections of cysts, varying in size from a pea to a marble, and separated from each other by septa of compressed renal or fibrous tissue. Either one, or frequently both, kidneys may be affected with what is sometimes termed *congenital cystic degeneration of the kidneys*. There is considerable enlargement of the organs, and during intra-uterine life they may attain a size so enormous as to render parturition extremely difficult and dangerous. The fetus is usually non-viable, though in mild cases the affection may be tolerated for some years after birth. The cystic fluid may be either clear or turbid, a reddish-yellow or a dark-brown in color, acid in reaction, and holds in solution urinary salts, blood, cholesterolin, and sometimes uric acid and urea. A single layer of flattened epithelial cells lines the cyst-walls. The cysts themselves seem to be dilatations of the renal tubules and of Bowman's capsules, due, in some instances, to an obliteration of the tubules of the papillæ or to stenosis of some portion of the urinary tract.

The cystic kidneys usually met with in adult life (acquired) are of several varieties: (1) One or perhaps a few cysts may be present, larger usually than those in the congenital cystic kidney, which seem to cause no interference with the normal renal functions. Sometimes a reddish-brown colloid material is contained in these cysts.

(2) Small and often quite minute cysts frequently accompany the chronic nephritic kidney that is small, contracted, and cirrhotic. These result from dilated tubules and capsules when the former are narrowed by the hyperplasia of fibrous tissue.

(3) Cystic kidneys in adults may have the pathologic characteristics of the congenital variety—a mere conglomeration of cysts containing a clear or colored serum or a cloudy, dark, thick, and colloid liquid. This condition is sometimes associated with similar cystic disease of the liver and spleen. It may be a late manifestation of mild congenital disease. The kidneys have been found converted into cysts in cases in which the presence of calculi (uric acid) in the tubules has probably started the cystic degeneration.

(4) Solitary cystic adenoma occurs rarely. It is in the form of a globular tumor projecting from the surface (usually the anterior) of the kidney. It may be as large as an orange, and may be enclosed in a dis-

tinct capsule. On section the mass is found to be composed of various-sized cysts separated by septa of fibrous tissue lined with cuboid or columnar epithelium. The remainder of the kidney appears to be quite healthy.

**Etiology.**—Cystic disease of the kidneys is either congenital or acquired. The former is probably commoner than the latter condition, and may persist for a while in extra-uterine life, while the acquired variety may be of unknown origin or secondary to chronic interstitial nephritis or to urinary calculi in the renal tubules. The direct cause of intra-uterine renal cysts is not definitely known, but they are probably developmental rather than pathologic, since other defects of embryonic growth are frequently associated with the disease.

**Symptoms.**—These may be absent in adults until the sudden development of uremia. Ordinarily, the clinical picture is similar to that of chronic interstitial nephritis. There is an increase in the quantity of urine, which is of low specific gravity. Slight albuminuria may be present. On *palpation* a large, rounded, and *sponge-like* mass may be felt in either hypochondrium or on both sides. Cardiac hypertrophy and increased arterial tension, as in chronic cirrhosis, are also frequently met with in cystic degeneration of the kidneys.

The **diagnosis** can only be made upon the presence of the above symptoms and the discovery of the clear physical signs of the tumor. It should be pointed out that a possible complication of perinephric abscess, due to rupture of one or more of the cysts (as has occurred—Osler), would of course render a diagnosis wellnigh impossible.

**Prognosis.**—Bilateral cystic disease of the kidney must eventually prove fatal, owing to the sudden onset of uremia or cardiac failure. Solitary cysts give a tolerably favorable outlook under proper surgical interference.

**Treatment.**—The unilocular cysts just referred to above may be removed, capsule and all, and the kidney sutured. Bilateral disease cannot be operated upon for obvious reasons; unilateral cystic degeneration may be treated by nephrectomy, with narrow chances of success.

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## NEW GROWTHS OF THE KIDNEY.

THE most common tumors of the kidney are those belonging to the class of adenomata (benign) and those that are either sarcomatous or carcinomatous (malignant).

**Adenomata** may be congenital or acquired. They grow in the cortex of the kidney in the form of small nodular masses, which in some cases may increase to a considerable size before any symptoms are produced. A cystic growth may be combined with adenoma (*cystic adenoma*), and *lymphadenoma* is also occasionally seen as a secondary growth. Other benign tumors that may affect the kidney are *angioma*, *fibroma*, and *lipoma*. Very large vascular adenomata may become malignant. Grawitz, Lubarsch, Kelly, and others have described a variety of tumor (*hypernephroma*) derived from aberrant adrenal tissue misplaced in the kidney.



**Sarcoma** and **carcinoma** may be either primary or secondary. *Sarcoma* is frequently congenital in origin, and may have an admixture of striped muscular tissue. The presence of the latter in the kidney points to developmental disturbances during embryonic life as the cause of a variety of tumor known as *rhabdomyoma*. Alveolar sarcoma is also met with.

*Renal carcinoma* is probably of less frequent occurrence than sarcoma; it may, however, be found in children as well as in aged persons, the two extremes of life. Carcinoma of the kidney is usually of the soft medullary or encephaloid variety. As a primary affection it probably originates in the renal tubules. Both sexes are subject to the disease. Secondary carcinoma of the kidney, although probably more frequent than the primary form, is seldom of clinical importance. Renal carcinoma may occur as a diffuse infiltration or in nodular masses, one kidney usually being affected in primary carcinoma. The tumor sometimes reaches an enormous size, and instances are recorded in which nearly the whole abdomen has been filled, and in which the growth weighed as much as 31 lbs. (14 kgms., Roberts). Rhabdomyomata do not, as a rule, attain a very large size, though sarcomata may grow quite large. Softening and hemorrhage within these malignant growths may occur. The pelvis of the kidney may be invaded, and metastatic areas may form in the liver or the lungs, though this occurs in the case of primary renal carcinoma less readily than from carcinoma in other organs. Metastatic growths arise most likely through involvement of the renal vein. The renal parenchyma is either partially or wholly destroyed, the pyramids being attacked later than the cortex.

**Symptoms.**—*Lumbar pain* on the affected side is often an early symptom, and may persist throughout the course of the disease. It may be paroxysmal, and be felt extending down the thigh, or it may be dull, dragging, and limited in character. Pain is not, however, a constant symptom in a certain proportion of the cases.

*Hematuria* may occur early or late, and often appears before any tumor is palpable. The blood may be in a fluid state or in clots, the latter not seldom taking the form of pelvic or urethral casts, the passage of which may give rise to colicky pains. Casts of the ureter sometimes resemble lumbricoid worms. The hemorrhage may be excessive and cause marked weakness and a symptomatic anemia, superadded to the cancerous anemia that is usually present; on the other hand, it may be so slight as to be discoverable only microscopically. It recurs at irregular intervals of days or weeks. Large clots may accumulate in the bladder and cause vesical irritability. The urine from the healthy kidney may be quite normal, and may be secured for observation by ureteral catheterization. Cancer-cells or tissue-fragments of the neoplasm very rarely appear in the urine, at least so as to be distinctly recognizable as such. *Anorexia, nausea and vomiting, progressive loss of flesh and strength*, increasing pallor, and the concomitant symptoms of the cancerous cachexia are seen to develop.

**Physical Signs.**—These may not be sufficient to reveal the presence of the tumor for some time after the above symptoms have been observed. The appearance of a palpable tumor in either flank is a definite aid to diagnosis. It is felt between the ribs and pelvis latero-anteriorly, and at first, when small and on the right side, it may be movable. Both

sarcoma and carcinoma of the kidney may assume enormous sizes. The tumor feels dense and hard (except rapidly-growing tumors, as encephaloid), either smooth or lobulated, and, when not too large, may retain the natural position and form of the kidney. The growth extends downward and inward, and in the very large malignant renal tumors of childhood the abdomen shows considerable enlargement, along with an abnormal pulsation and a prominence of the veins. Usually the tumor does not move with respiration. Percussion gives dulness over the mass, although in small and moderately large tumors the overlying colon may cause a tympanitic note to be heard. Neighboring organs, as the liver and spleen, may be found by palpation and percussion to be displaced by the renal tumor.

**Diagnosis.**—The presence of a tumor, when not too large and distinctly occupying the lumbar and lower lateral abdominal region, together with hematuria, pain of a local nature, and progressive failure of nutrition, may be looked upon as diagnostic of a malignant type of renal tumor. The relation of the colon to the tumor and immovability of the latter during respiration are also diagnostic. When the tumor is very large and adhesions have formed, as in cancerous kidney, it may be mistaken for other conditions.

**Differential Diagnosis.**—Affections such as hydronephrosis, pyonephrosis, cystic kidney, hydatids, ovarian, splenic, and hepatic tumors, and (particularly in children) retroperitoneal sarcoma must be differentiated from renal growths. Careful bimanual palpation will aid in the diagnosis, but the exclusion of other lumbar enlargements must be made by close attention to the history and to the development and course of the symptoms. Hematuria alone, in aged persons, is suggestive of carcinoma when no tangible cause for the presence of the blood is at hand. Hepatic and splenic tumors are usually movable during deep breathing, whilst renal tumors are not so. In cases of hepatic growths also the area of dulness extends higher, whilst in renal growths on the right side a tympanitic area generally lies between the liver and the tumor. The characteristic notch and edge of the spleen, and the absence of the overlying colon-tympany, are points that distinguish splenic enlargements from those of the left kidney. Pelvic growths (ovarian and uterine) enlarge from below upward, and are readily detected by vaginal examination. In children Löbstein's cancer (retroperitoneal sarcoma), if very large, is easily mistaken for a renal tumor, except that it is usually more centrally situated and more firmly fixed.

**Prognosis and Treatment.**—The termination in cases of renal carcinoma is inevitably fatal, and children succumb more quickly than adults. The disease may last from a few months to sometimes a year or two.

If the kidney be removed while the growth is still small, the prognosis is fairly good; but if large or if metastatic tumors have formed, the prognosis is always bad. Bloch warmly advocates in some cases the removal of small sections of kidney-substance, to avert the necessity of a nephrectomy by proving the non-malignancy of the growth. The *treatment*, aside from early surgical measures, is entirely symptomatic and supportive, and obviously it is unsuccessful. Renal colic, excessive hematuria, and a gradually lowered vitality may be met by the use of palliatives, tonics, and by nutritious and easily digestible diet. Nuclein may be tried hypodermically or by the mouth.

## II. DISEASES OF THE BLADDER.

## CYSTITIS.

**Definition.**—Inflammation of the mucous membrane of the bladder. It may be either *acute* or *chronic*, the latter being clinically the much more frequent condition.

## ACUTE CYSTITIS.

**Pathology.**—Cystoscopic examination performed according to Pawlik's or Kelly's method, hereafter to be described, reveals an intensely hyperemic condition of the vesical mucosa, which is puffy, edematous, and of a bright-red color; this may be more intense at points, especially in the vicinity of the trigone. The membrane is bathed in a thick, tenacious muco-pus, and here and there may be noted denuded areas, and the exfoliated epithelium often hanging in shreds from the bladder-wall; overlying these denuded patches hemorrhagic effusions may be observed. In the severer grades of the disease the intense general hyperemia causes a disappearance of the blood-vessels that are to be seen in the normal condition. Occasionally small patches of ulceration, due to abscess-formation (*phlegmonous cystitis*), may be observed, and in rare and fatal instances the entire bladder-wall is involved in a necrotic process.

**Etiology.**—Cases of acute cystitis may be grouped according to their origin into four main classes, as follows:

(1) **Catarrhal.**—Like other mucosæ, the vesical epithelium is very responsive to systemic circulatory disturbances. Thus, sudden exposure to extremes of cold or heat or violent atmospheric changes, thereby abruptly suppressing the action of the skin, may be potent influences in the etiology of the disease. An intense acute catarrhal inflammation may follow retention of the urine in the bladder, with or without its subsequent decomposition; it may also be the result of pressure from an enlarged prostate or other tumor, and may follow cystocele, urethral stricture, or paresis of the bladder-wall. In simple over-distention of the bladder, with the accumulation of a gallon (4 liters) or more of urine, the so-called acute *exfoliative cystitis* may result, in which the entire mucous membrane of the bladder may be shed, and the patient shortly manifest all the symptoms of grave uremic intoxication. The prolonged retention of urine is followed by decomposition of the fluid, and this by its irritant action always excites a cystitis that soon assumes the chronic type.

(2) **Septic.**—This may result either from the direct introduction of pus-producing germs into the bladder or from the systemic transmission of these micro-organisms to the organ. This is known as the *bacterial origin* of cystitis. Under the first class may be mentioned the passage of a dirty catheter or sound; this is a cause of cystitis in puerperal women, and in men who are the subjects of minor grades of urethral stricture, and who have been subjected to gradual dilatation by means of bougies. *Gonorrheal cystitis* is also to be included under this heading. There is a condition known as *febrile cystitis*, which constitutes the second class of septic cases. This comprises the vesical in-



flammation that is present in the various febrile conditions, and which is probably a direct result of the presence in the urine of the causal bacilli or their toxins (Fitz). Thus, in all of the infectious diseases and fevers (typhoid and the other exanthemata, rheumatism, diphtheria, tuberculosis) there is noted a cystitis of varying degrees of severity that can be accounted for only by the local irritant action of the specific germ of the associated disease, or its eliminating toxins. The so-called *gouty cystitis*, which is often present in lithemic individuals, and which is due to the irritating and concentrated urine, may also be here included.

(3) **Toxic.**—Certain drugs when introduced into the system manifest an irritant action upon the vesical mucosa, and promptly excite a severe grade of acute cystitis. Prominent among these may be mentioned cantharides and other irritants of the urinary tract—cubebæ, copaiba, and sinapis. Workers in coal-tar dye-stuffs are sometimes affected with acute cystitis.

(4) **Traumatic.**—Traumatic inflammation of the bladder follows the improper and careless use of the catheter, sound, or other instrument; the presence in the bladder of calculi or other foreign bodies; and the pressure of the fetus in parturition, or of large masses of impacted feces.

(5) **From Adjacent Inflammation.**—Irritation with consecutive inflammation may result from the extension of an inflammatory process from surrounding structures either by continuity or contiguity of tissue. Thus, a cystitis may follow a urethritis—gonorrheal or otherwise; it may result from an extension downward of a ureteritis, or it may be consequent upon a vaginitis, a malignant neoplasm of an adjacent viscus, a salpingitis, pelvic peritonitis, or pelvic abscess in the immediate vicinity of the bladder, as in the vesico-uterine pouch, the inflammation extending by an involvement of contiguous tissue.

**Symptoms.**—The symptoms of acute cystitis are very marked. *Pain, vesical irritability, vesical and rectal tenesmus, frequency of micturition, fever, and urinary changes* are all pronounced. Prominent among these is *pain*, which may be most intense and is the earliest and most persistent manifestation of the disease. Its seat is the suprapubic region, whence it may radiate to the sacral region, the perineum, the end of the penis, or the upper portion of the thighs; it is most constant, but is worst just before micturition, by which it may be alleviated. It is considerably relieved by the recumbent posture, and is aggravated by pressure over the bladder. As the inflammatory process diminishes the pain gradually disappears, and the entire attack may subside in a few days or a week.

With the pain, and probably ranking second in severity, is the *rectal and vesical tenesmus*, or *strangury*. There is an almost constant desire to urinate, the patient sitting upon the urinal, it may be, for hours. The *urine* may be opaque or highly-colored. It is often bloody (in very acute cases the vesical contents may consist of a small quantity of pure blood only), is of a specific gravity varying from 1005 to 1030 (in the febrile cases), and contains pus-corpuscles in abundance, mucous flakes in large quantities, shreds of disintegrated and exfoliated epithelium (bladder); also numerous micro-organisms (streptococci, staphylococci, gono-

cocci, proteus vulgaris, bacilli of tuberculosis, and very commonly the bacillus coli communis); fungous mycelial threads and yeast-cells have even been found in certain cases (*mycotic cystitis*). Its reaction may be either acid or alkaline; if alkaline, it contains ammonium urate, amorphous phosphates, and triple phosphates (crystalline) as a rule. More or less albumin will be noted, and on standing a dense sediment forms in the bottom of the flask, composed of all the foregoing substances, as shown by chemical and microscopic examination. The total quantity of urine voided in the twenty-four hours may be normal in amount or even slightly in excess of the normal. On the other hand, if exfoliation of the mucous membrane takes place, there may occur partial or even total suppression of the urine. *Fever*, with or without an initial rigor, persists throughout the attack, but is not of a severe type, save in the septic and malignant (diphtheritic) forms of the disease, when it may reach 103°–105° F. (39.4°–40.5° C.).

*Abscesses* may form, and betray themselves by localized pain, tenderness, and, in some cases, by a circumscribed induration. These may rupture into the bladder, followed by the free escape of pus from the urethra and by relief (temporary as a rule) from urgent symptoms, or they may spread to the peritoneum and induce peritonitis, which, if not promptly treated by surgical measures, may prove fatal by gradual asthenia.

In the variety associated with extreme exfoliation of the vesical mucosa *grave uremic manifestations* follow. These include all the features of the typhoid state (dry, brown tongue, mild delirium, nervous and muscular twitching; headache; gastric disturbances; and coma). There is also some degree of malaise and anorexia.

It must not be forgotten that acute cystitis may represent an acute exacerbation in the chronic form, and at times may assume a severe type of the disease.

**Diagnosis.**—Cystitis should be readily recognized from the history of the case and the frequency of the two almost pathognomonic symptoms—suprapubic pain and vesical tenesmus. An examination of the urine will reveal the characteristic clinical features. Cystitis may be confounded with *acute nephritis* or *pyelo-nephritis*, but a careful study of the clinical manifestations and, if need be, the catheterization of the ureters after vesical irrigation, will reveal the true condition. The presence of tube-casts in the urine would indicate renal involvement. The percentage of albumin is usually much larger in nephritis than in irritability of the bladder. The differentiation between cystitis and vesical irritability will be noted under the latter condition.

The **prognosis** of the milder grades of cystitis is good; the septic and malignant (diphtheritic) cases offer a much graver outlook. Extension of the process upward toward the kidneys is always serious.

**Treatment.**—The treatment of acute cystitis includes prophylactic, hygienic, and medicinal measures.

**Prophylactic.**—Most important is the prevention of the disease, and this includes, in addition to the usual care of the body, the observance of thorough asepsis whenever it becomes obligatory to introduce an instrument (catheter, sound) into the bladder.

**Hygienic.**—The cause of the disease, if evident (calculus, external pressure), should be sought and removed. The patient should at once be placed absolutely at rest in the recumbent posture. The value of this injunction will be most clearly understood when it is stated that in the erect position the intra-vesical pressure is three times that in the dorsal position. The simple observance of this law will do much toward relieving the sufferings of the patient. The *diet* must be regulated, and all irritating, highly seasoned articles of food must be interdicted. Alcohol in any form is prohibited. If it can be enforced, during the early stages of the disease an absolute milk diet will be most beneficial. The patient should be instructed to drink freely of water and other diluent drinks, whereby an internal irrigation of the bladder may be secured and much of the irritating substance removed. The free action of the skin may be secured by friction and warm bathing.

**Medicinal.**—The drugs to be employed are the saline laxatives and the various mild diuretics and urinary alterants. The reaction of the urine will indicate the variety of alterant to be employed. If it be acid, alkaline waters are serviceable, as the soda-preparations, Vichy, or the potassium salts. In alkaline conditions of the urine probably the most valuable drugs are benzoic and boracic acid and salol. Benzoic acid is best administered in the form of ammonium benzoate, which may be given in 10-grain (0.648) doses thrice daily in the compound infusion of buchu, or in uva ursi. Hot applications and hot local bathing (sitz-baths) will do much to relieve the pain and tenesmus; if these be severe, a rectal suppository of opium and belladonna or an enema of chloral hydrate will generally give prompt relief. Tincture of cannabis indica, administered internally, may answer if opium be contraindicated. Under such a course as the preceding a cure may be expected within eight or ten days. It is prudent to advise the patients to wear flannel or silk binders over the abdomen, to avoid chilling of the surface and subsequent acute attacks.

#### CHRONIC CYSTITIS.

**Pathology.**—The vesical mucosa is not so hyperemic as in the acute variety, but is of a peculiar muddy or grayish-blue (slate) color, dotted here and there with patches of erosion or of actual ulceration. The muco-pus that bathes its surface is not so apt to be hemorrhagic as in the acute form of the disease, although slight hemorrhages may and do occur. Owing to the slow course and long duration of the disease there follows an immense thickening of the bladder-wall from hyperplasia of its constituents, conjoined with more or less edema of the tissues. The result is a contraction of the wall with a proportionate diminution in the vesical capacity. The mucosa may become, as it were, polypoid in spots, and there may follow obliteration or partial obstruction of the ureteral orifices, with consequent dilatation of the ureters and renal pelves from a damming back of the secretion. The urinary changes are about as in the acute form, save that the reaction is always alkaline and the amount of mucus and pus is proportionately greater.

**Etiology.**—Chronic inflammation of the bladder may be the result of a neglected or oft-repeated acute attack. It may occur from the persistent action of an exciting cause, as the presence of some irritating



substance (calculus) in the bladder, or of some excitant external to that viscus, as a localized inflammation or a displaced uterus. Again, the inflammation of the bladder may be of a chronic nature from the beginning; especially is this true of the tuberculous variety and of that due to neoplasms of the organ.

The **symptoms** and **diagnosis** differ but slightly from those of acute cystitis. It may, however, be pointed out that the pain and tenesmus are less intense. Oppositely, the *amount of albumin* in the urine is comparatively large. The same remark applies to the *quantity of mucus* and *pus* (*vide* Pathology); indeed, the last-named ingredient often forms a thick gelatinous mass in the standing urine that tends to adhere to the receptacle. Chronic cystitis is accompanied by debility and emaciation, which, however, are of slow development.

The **prognosis** is always serious, and the course of the disease is at the best protracted.

**Treatment.**—Very generally, the treatment set down for the acute disease will not answer in the chronic form. Undoubtedly, there will follow more or less amelioration of the symptoms, but the tendency is toward a prolonged chronicity. In such cases, after the removal of the ascertainable causes so far as practicable, we are compelled to resort to local treatment of the bladder. This includes—(1) Vesical irrigation; (2) Topical applications; (3) Permanent drainage of the bladder.

*Vesical irrigation* is secured by means of an aseptic soft-rubber catheter which is connected with a graduated glass funnel: a siphonage is produced by the alternate elevation and depression of the funnel, which contains the irrigating fluid. The latter may consist of plain sterilized (boiled) water, sterile normal salt-solution (40–60 gr. to the pint—2.59–4.0 per  $\frac{1}{2}$  liter), or a weak solution of mercuric chlorid (1: 50,000–100,000). The irrigation should be done slowly, and not more than twice or thrice daily in severe cases, and much less frequently in ordinary cases, according to the exigencies of the condition.

*Vesical medication* may be secured by means of the funnel after irrigation, the medicating substances being dissolved in a pint of water and allowed to flow slowly in and out of the bladder. The drugs that may be used in this manner are silver nitrate or zinc sulphate (1–5 gr. to the ounce—0.0648–0.324 to 32.0) or a saturated solution of boric acid. If the salts of zinc or silver are used, not more than an ounce of the solution should be allowed to enter the bladder, and much less than this amount will generally suffice. In cases in which there exist patches of ulceration the application must be made directly to these areas through the endoscope or cystoscope (Pawlik, Kelly). In women this may be readily done by placing the patient in the exaggerated lithotomy or knee-chest posture, dilating the urethra, and introducing the cystoscope, through which a reflected light is thrown upon the distended bladder-wall. Stronger solutions may now be employed, as silver nitrate, 20–30 gr. (1.29–1.94) to the ounce. This application should be followed by a slight irrigation of the bladder.

When this local medication fails to effect a cure, permanent drainage of the bladder must be secured—in the male by a suprapubic or perineal incision, and in the female by the establishment of a vesico-vaginal fis-

tula. This places the bladder absolutely at rest, and gives the inflamed mucosa a chance to heal under proper medication.

As to internal remedies, various agents that possess a local stimulating effect upon the genito-urinary tract are advised by most authors, but I think little is to be gained from their employment as compared with the results achievable from topical treatment. Most efficacious among internal remedies are—oil of sandalwood, terebene, pichi, buchu (fluid extract), and the oil of copaiba. If disinfection of the bladder *in loco* is not practicable, antiseptics should be given internally, combined with those stated above. Salol and potassium chlorate are excellent for this purpose.

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### NEOPLASMS OF THE BLADDER.

PRIMARY new-growths of the bladder are exceedingly rare, occurring, however, with greater frequency in males in about the proportion of 3 to 1; they may be either benign or malignant. On the other hand, secondary neoplasmata, particularly carcinomata, are relatively common. The most frequent variety of new-growth encountered is carcinoma, particularly the so-called villous or papillomatous carcinoma, Williams<sup>1</sup> finding in 20 women affected with bladder-tumor, carcinoma in 16. Other growths are sarcomatous, fibromatous, cystic, and papillomatous in nature.

The **symptoms** are the same for all varieties, and include, first and most commonly, *hemorrhage* (which is both persistent and free), together with pain, frequency of micturition, and occasionally the discharge of detached fragments of the growth. In carcinomatous cases of advanced standing cachexia will be marked. Examination by means of the cystoscope will reveal the nature of the complaint. In the case of secondary growths the primary tumor may often be detected.

The **prognosis**, of course, will depend upon the nature of the growth.

The **treatment** is purely surgical, and comprises enucleation of the tumor either by means of the snare, or after a vesical section.

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### VESICAL HEMORRHAGE.

(*Vesical Hemorrhoids.*)

HEMORRHAGE of the bladder has been mentioned as a symptom of various affections, both general and local, among the former being leukemia and malarial hematuria, and among the latter nephrolithiasis and tuberculosis and carcinoma of the bladder. It is also a prominent manifestation in stone in the bladder, and not infrequently appears in pregnancy (late). Independently of the operation of all of the above-mentioned etiologic factors, hemorrhage has been known to occur from the bladder, and recent precise methods of exploring the viscus (endoscopic

<sup>1</sup> *Brit. Med. Journ.*, 1889.

examination) have shown it to be due to a hemorrhoidal state of the vessels. The hemorrhage may be profuse, and, rarely, even fatal in its effects.

The **diagnosis** is based in part upon the absence of the more obvious causes of hematuria and the presence of free bleedings, but chiefly upon the result of a careful cystoscopic exploration of the bladder.

The **prognosis**, so far as my experience extends, is eminently favorable, though a few fatal cases have been reported.

**Treatment.**—This is mainly local. The bladder may be irrigated with an astringent solution (1 per cent. tannic acid,  $\frac{1}{2}$  per cent. alum), and this may be alternated with an antiseptic solution (3 per cent. boric acid, 1 per cent. salicylic acid). I have recently observed a case in which recovery followed the internal admission of the extract. hamamelis fluid. (5j-4.0), *t. i. d.*

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## NEUROSES OF THE BLADDER.

### IRRITABILITY OF THE BLADDER.

**Definition.**—By this term is meant a condition of the bladder in which there exists an hyperesthesia of the organ, especially of the neck—that portion surrounding the urethral and ureteral orifices (*vesical trigone*)—without the presence of any tangible cause therefor. This must be distinguished from the irritability that is associated with true organic disease of the bladder itself, as in the presence of calculi, tumors, or fissure of the neck, or with disease of the surrounding structures.

**Pathology.**—There are no pathologic features to be noted. A cystoscopic examination of the bladder may reveal a slight increase in the vascularity of the mucous membrane, but the condition, in most instances at least, must be regarded as a true neurosis. The condition of irritable bladder in women, which has previously been held to be a purely functional derangement, is now regarded by Dacheux and Zuckerkandl as a localized hyperemia, especially at the *bas fond*, and less often at the beginning of the urethra.<sup>1</sup>

**Etiology.**—While in many instances no well-defined causal relations can be determined, it is very generally true that the patients who are the subjects of vesical irritability are individuals of a neurotic temperament, very often manifesting strong hysteric tendencies. They present the characteristic features of this unfortunate group. They are generally ill-nourished, fretful, irritable, peevish, suffering almost constantly from vague neuralgic attacks in different portions of the body (cephalalgia, tic douloureux, lumbo-sacral pain), and in a chronic condition of physical prostration. Frequently they eventually develop a true hypochondriasis or melancholia. In others there may be found a history of extreme mental and physical tire, overwork, business anxiety, over-indulgence in venery, menstrual irregularity, dysmenorrhea, ovarian or uterine disorders, long-continued gastro-intestinal disturbance (dyspepsia), improper hy-

<sup>1</sup> *The American Year-Book of Medicine and Surgery*, 1897, p. 576.



gienic surroundings, improper regimen, indulgence in late hours, and a general lack of will-power. It must, however, be remembered that subjects of chronic malarial intoxication very often manifest all the symptoms of vesical irritability, marked, it may be, by a feature of more or less periodicity. This has been termed by some *malarial fever of the urethra and bladder*. Lithemic individuals also are very prone to develop a pronounced vesical irritability, the affection in them probably resulting from the local action of the highly concentrated and irritating urine. The condition must commonly, however, be regarded as belonging essentially to the large group of neuroses.

In a certain percentage of cases the bladder-trouble is a reflex manifestation of some disease of an adjacent organ, as the urethra, ureter, vagina, rectum, anus, or the internal organs of generation. These are not, however, to be looked upon as cases of true neurotic vesical irritability.

**Symptoms.**—The symptoms of irritable bladder are mainly extreme painfulness and *frequency of micturition*, associated with marked *vesical and rectal tenesmus*. The dysuria is not always or altogether relieved by micturition; indeed, the pain may be just as severe, or even worse after, than before, the voiding of the urine. Especially is this true when there coexists a more or less spasmodic muscular action of the bladder-walls, the hypersensitive mucosa then being squeezed, and the patient suffering at times to such an extent as to be thrown almost into a state of collapse. There is usually a sense of weight or pressure in the pubic region, which is largely relieved when the patient assumes the recumbent posture. Urination is often performed spasmodically, or there may be a *spasm* of the urethra and neck of the bladder resulting in an utter inability to perform the act. The *urine* may be normal in appearance and amount. Very often it is increased in quantity (*hysterical polyuria*), and at times the opposite may be true and more or less suppression be noted. In lithemic cases the urinary characteristics already mentioned under that condition will be present (*vide* p. 401).

**Diagnosis.**—Very frequently will simple vesical irritability be confounded with true cystitis. The points of differentiation, however, are as follows:

#### IRRITABLE BLADDER.

#### CYSTITIS.

The patient is of a neurotic temperament, and generally gives no history of organic bladder-disease nor of operations upon the bladder.

Pain is severe, and often worse after micturition.

The constitutional symptoms are those of nervous depression.

Never results fatally.

The urine does not present any marked alteration in its physical or chemical qualities. It may show hyperacidity, or extreme concentration, or dilution.

The appearance of the mucosa is negative in true neurosis.

The duration is always protracted.

May occur in any individual, irrespective of temperament. It frequently follows catheterization, sounding, or other traumatism.

The pain is usually much relieved by micturition.

The constitutional symptoms are not marked, save in grave cases.

May result fatally.

There are always present marked and characteristic alterations in the physical and chemical qualities of the urine.

Cystoscopic exploration reveals the angry and diseased mucosa, and may show the cause (calculus, tumor).

The duration of acute attacks may be short.

**Prognosis.**—Good as regards life; doubtful as regards the ultimate cure of the patient.

**Treatment.**—Since the condition is largely one of neurotic origin, the attention of the physician must be directed mainly toward a betterment of the state of the nervous system. Absolute rest, physical and mental, must be insisted upon, and the patient must be subjected to a course of strict moral suasion whenever this may be deemed necessary. Any cause of reflex irritation must be removed, and a careful search should be instituted for some such condition as cervical stenosis, uterine displacements, anal fissure, hemorrhoids, stricture of the rectum, vaginitis, urethritis, tuberculous infection of Skene's glands of the urethra, chronic gastro-intestinal catarrh, and the like. The habits of the patient must be inquired into, and late hours, the eating of improper and unwholesome articles of food, masturbation, or the reading of sensational and trashy literature corrected. In many instances the pronounced neurasthenic condition demands a course, more or less protracted, of the Weir-Mitchell rest-treatment (*vide* Neurasthenia, p. 1177). The urine should be carefully examined for lithemic and other pathologic features, and by an appropriate course of treatment it should be rendered as bland and unirritating as possible. Large draughts of diluent drinks may be of benefit, and if these be combined with the prolonged administration of nerve-sedatives and antispasmodics, a marked amelioration of the patient's condition may be secured. In cases associated with spasmodic muscular contraction it may become necessary to employ an occasional suppository of opium and belladonna, or an enema of chloral hydrate. Change of air and scene, regulation of the diet, the institution of a proper course of gymnastics, mental and physical, and the observance of a happy and cheerful atmosphere will generally do much to improve the patient's condition. The administration of tonics (strychnin, iron) and the prevention of constipation are very essential. Especially must it be remembered that in all these cases of simple vesical irritability physical exploration of the bladder is absolutely contraindicated. The patient's mind must be directed away from the bladder in order to secure good results.

#### NEUROSES OF MICTURITION.

1. **Incontinence of Urine** (*Enuresis*).—An inability to retain the urine. This may arise from a number of causes. Frequently it is the result of some lesion of the spinal cord involving the sphincteric center of the bladder; this is known as *paralytic incontinence*, and is to be recognized by a constant dribbling, alternating with spurts of urine when voluntary or involuntary muscular action is brought into play, as in the act of coughing, sneezing, or bending forward of the body. It may be the result of a general bodily weakness or after prostrating diseases (typhoid, late stages of pulmonary tuberculosis). Again, it may result from some local condition in the bladder or urethra. Here may be mentioned paralysis of the urethra from over-dilatation or from traumatism, or that due to pressure of the fetal head in a prolonged labor; imperfect vesical innervation; over-distention of the bladder, producing a paresis of its walls; or from some temporary obstruction at the urethra or base of the bladder, such as a tumor or a sharply retroflexed uterus.

It may be a result of over-distention of the bladder, with partial paralysis of the sphincter, the bladder remaining overfilled, while there is a constant escape of a few drops of urine (*incontinence of retention*). It may follow some local causes of irritation, as the presence of vesical calculi, pressure from an anteflexed uterus upon the fundus of the bladder, cystitis, and parasites. The condition known as *spasmodic incontinence* is that due to an over-action of the compressor muscle of the bladder, as a consequence of which there is a diminution of the vesical capacity, the urine being forcibly and involuntarily ejected at irregular intervals. Finally, *nocturnal enuresis* is that variety which is so common in young, delicate, and often neurotic children: this is usually noticed in the early hours of sleep, and is often the result of some local irritation acting upon a hypersensitive organism, such as the presence of ascarides, an elongated prepuce, contraction of the urethral meatus, or masturbation. Nocturnal incontinence may be a manifestation of nocturnal epilepsy or of incipient cerebral or spinal disease (Fitz). The constant escape of urine in the paretic cases is apt to result in extensive excoriation of the parts.

The *treatment* varies according to the cause. The enuresis of children, if left alone, will eventually cure itself as the age and strength of the patient increases, though obvious exciting causes, if present, should be removed if not impracticable. Good hygiene, systematic evacuation of the bladder, elevation of the hips on a pillow in bed, plenty of out-of-door exercise, a change to the seashore or mountains, an abundance of suitable and strengthening food with a minimum of water, and the administration of tonics (iron, cod-liver oil, and strychnin), will generally effect a cure. The fluid extract of *rhus aromatica* in 5 to 15 drop doses, thrice daily, has been very beneficial in children. Excellent results often follow the administration of minute doses of atropin or tincture of belladonna. A favorite formula of my own in cases possessing a hypersensitive nervous organization has long been as follows:

R̄. Tr. belladonnæ,	3ss-j ( 2.0-4.0);
Sodii brom.,	3ij ( 8.0);
Ac. hydrobrom. dil.,	3ijss (10.0);
Ext. ergotæ fl.,	3ij ( 8.0);
Glycerini,	3j ( 4.0);
Elix. simplicis, q. s. ad	3iv (128.0).

M. et Sig. 3j (4.0) three or four times a day for a child of five years.

In very delicate or feeble children suffering from enuresis I substitute a motor tonic and stimulant (tr. nucis vom.) for the bromids or nerve-sedatives.

Spasmodic action of the vesical compressor may be relieved by the cautious use of the motor depressants, while its converse, paresis, demands the exhibition of full doses of strychnin or tincture of nux vomica. The judicious and careful use of the catheter, followed by the administration of strychnin, will promptly effect a cure in the incontinence of retention. Any local cause of vesical irritation must be removed. Galvanism in the paretic cases, applied both to the bladder and to the urethra, may be of service, and in the female Sänger suggests massage



of the urethra. Should excoriation occur, bland ointments, as of zinc oxid and lanolin, should be used.

**2. Retention.**—Nervous retention of the urine is occasionally encountered in hysteric and highly neurotic individuals. Its most common manifestation is an inability to urinate in the presence of others. It is also occasionally noted after childbirth, when it may be due to nervous reaction, to edema and tortuosity of the urethra, or to a temporary inability of the bladder-walls to contract upon their contents, thereby permitting a longer retention of the vesical contents, and even favoring over-distention of the organ. If the urine be allowed to remain for too long a period in the bladder, fermentative changes follow and a secondary cystitis will result. Under these circumstances an exfoliation of a portion or even of the entire bladder-epithelium may be noted.

The *treatment* consists in the administration of strychnin and other nerve-tonics, in building up the general constitution, and in affording a change of air and recreation. In that variety following childbirth the patient should be urged to make voluntary efforts at micturition, and these may be seconded by the firm application of an abdominal binder and compress, or of hot, moist flannel cloths, kept up for twenty minutes or a half hour. The sound of running water, as when pouring water from a pitcher into the basin, often causes a contraction of the bladder and excites the flow of urine. It may become necessary, the foregoing methods failing, to resort to catheterization, the usual antiseptic precautions being observed.

## PART VIII.

# DISEASES OF THE NERVOUS SYSTEM.

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THE central nervous system is generally divided into two parts—the brain and the cord. The *brain* consists of the cerebral hemispheres, the basal ganglia, the pons, the cerebellum, and the medulla. The cerebral hemispheres are joined together by the corpus callosum and the anterior and posterior commissures. They are united to the pons by the crura cerebri, and the pons is continuous with the medulla, which in turn is continuous with the spinal cord. The surface of the cerebral hemispheres is divided by sulci or fissures into various regions, known as the frontal, parietal, temporo-sphenoidal, and occipital lobes. The superior longitudinal fissure separates the two convolutions; the fissure of Sylvius is between the frontal and parietal lobes above and the temporo-sphenoidal lobe below. The fissure of Rolando divides the frontal from the parietal lobe, and the parieto-occipital fissure the latter from the occipital lobe. The continuation of the last-named fissure upon the median surface forms the upper boundary of the cuneus, the lower boundary of which is the calcarine fissure. The hippocampal fissure separates the fascia dentata from the hippocampal gyrus, and by its extension inward produces an elevation in the lateral ventricle known as the hippocampus major. Each lobe is subdivided by secondary fissures into a number of lobules. The topography of the hemispheres is important because it is now possible to map out with considerable accuracy the regions in which various motor impulses originate, and with less accuracy the regions in which various sensory phenomena are perceived. The accompanying diagrams illustrate, more satisfactorily than could any description, the regions that have been hitherto determined. There is some discussion in regard to the degree of individuality of these centers, but the weight of evidence inclines to the belief that they are not sharply delimited. Ordinarily speaking, one side of the brain innervates the opposite side of the body; but certain parts, as the muscles of the trunk, appear to receive impulses simultaneously from both hemispheres, and other functions seem to be accomplished exclusively upon one side; thus motor speech is ordinarily disturbed only when the lesion is in the left hemisphere.

The central nervous system is composed practically of two elements—the neuroglia, or supporting substance, and the neurons. The neuroglia consists of round cells with radiating processes, lying in the





other parts, especially the axis-cylinder. It probably also generates the nervous impulses. The protoplasmic processes may have nutritive functions, or serve to conduct impulses to the cells (cellipetal). The axis-cylinder conducts impulses from the cell (cellifugal), except in the case of the peripheral process of the cells of the spinal ganglion.<sup>1</sup> A short distance from the cell the axis-cylinder is enveloped by the myelin-sheath, giving rise to the nerve-fiber, and when aggregated together these fibers form the white matter of the nervous system.

It has been possible to trace more or less accurately the course of many of the groups or systems of fibers. These exist because cells

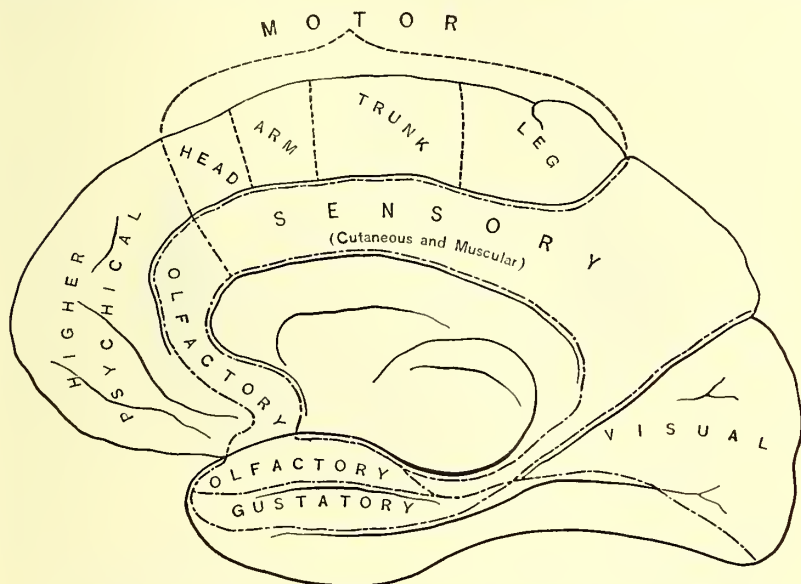


FIG. 68.—Zones and centers of the mesial aspect of the human cerebrum (Mills).

having the same functions are usually grouped together, forming centers or ganglia, and the fibers from these, taking the same course, form a bundle. Three classes are recognized: (1) fibers wholly within one hemisphere, *fibræ propriae*, uniting adjacent convolutions, and long association-fibers, uniting different lobes; (2) fibers passing from one hemisphere to the other, commissural fibers; (3) fibers passing from the cerebrum to the other parts of the central nervous system, the projection-fibers, forming the *corona radiata*.

The columns or tracts that have been mapped out in the cord may be seen in the accompanying diagram (Fig. 69). In the antero-lateral portion are found the anterior or uncrossed pyramidal column, the antero-lateral column of Gowers, the cerebellar column, and the crossed pyramidal column. In the posterior region are the columns of Goll and Burdach. The rest of the white matter forms the so-called ground-bundles.

In the area comprising the anterior and lateral columns both ascending and descending fibers are found.

<sup>1</sup> Lenhossek has suggested that this is a modified protoplasmic process.

The columns that transmit *ascending impulses* are—1. The direct lateral cerebellar column. 2. The antero-lateral ascending column of Gowers. 3. The antero-lateral ground-bundle or fundamental column. 4. The columns of Goll and Burdach. *Descending impulses* are transmitted chiefly by the direct and crossed pyramidal tracts and the antero-lateral descending tract. The direct lateral cerebellar tract of Flechsig takes origin in the cells of the column of Clarke, and first appears in the lower dorsal region, and passes through the restiform body to the cerebellum. Gowers' tract, or the antero-lateral ascending column, is first seen in the lumbar cord, and arises from some of the cells of the posterior horn. It then crosses to the other side of the cord through the posterior commissure and terminates in the region of the lateral nucleus.

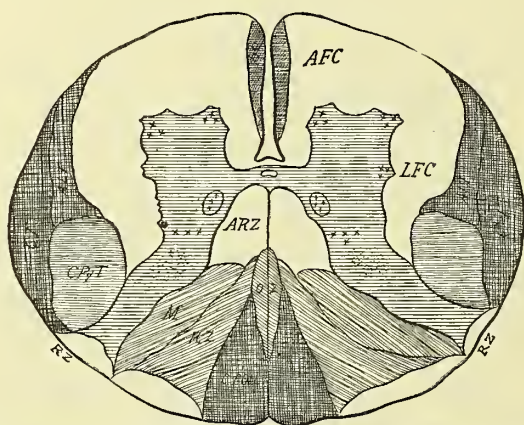


FIG. 69.—Section of spinal cord (after Dana), showing complete subdivision of white columns into—

Anterior columns.	{	DPy, direct pyramidal tract. AFC, anterior fundamental column.	Lateral columns.	{	LFC, lateral fundamental column. LL, lateral limiting layer. CPyT, crossed pyramidal tract. CT, direct cerebellar tract. ALT, antero-lateral ascending tract.
Posterior columns.	{	Column of Goll. Column of Burdach. RZ, rim-zone, or Lissauer's column.		{	ARZ, anterior root-zone. MRZ, middle root-zone. OZ, oval zone. PRZ, posterior root-zone.

The direct and crossed pyramidal columns constitute the great motor path by which fibers descend from the cortex and end in the motor nuclei of the cranial and spinal nerves—in the latter case in the multipolar ganglion-cells of the anterior horns. Their origin is in the motor region of the cerebral cortex—*i. e.* the ascending frontal and parietal regions, the paracentral lobule, and the posterior part of the inferior frontal convolution; they then approach one another, as do fibers from all parts of the cerebral cortex (known collectively as the *corona radiata*), to enter the internal capsule. This may be described as a wedge, bounded in front and to the inner side by the caudate nucleus and the optic thalamus, and on the outer side by the lenticular nucleus.

All of the fibers of the corona radiata do not pass through the internal capsule, some being lost in the gray matter of the basal ganglia, while

FIG. 70.

FIG. 70.

FIG. 71.—1, Motor centers for the lower extremities; 1<sup>1</sup>, motor centers for the upper extremities; 2, motor centers for the nerves of the face; 3, 4, 5, lateral pyramidal tract (red); 6, 7, 8, anterior pyramidal tract (green); *py*, pyramids (red); *col* (red and green), collateral fibers leading to gray substance. The Roman numerals (III, IV, etc.) indicate the nuclei, and correspond with the numbers of the cerebral nerves; the letters (*g*, *h*, etc.) represent the points of decussation and the names of the individual nerves.





others take origin in the ganglia. The angle of the internal capsule is known as the genu or knee, the part anterior to it as the anterior limb, and the posterior portion as the posterior limb. Through the anterior limb pass the fibers from the frontal region; in the region of the genu are the fibers for the muscles of the face and tongue; and in the posterior limb, the motor fibers to the extremities, also the sensory or tegmental fibers, and at its posterior end the fibers of the optic radiation.

The crusta consists of fibers that pass through the pons and enter the medulla, constituting its pyramidal tracts.

The tegmental fibers are continuous through the longitudinal fibers of the pons with those derived from the *formatio reticularis* of the medulla. This is formed by fibers from the superior cerebellar peduncles, the olivary body, and the posterior and lateral columns of the cord, which are reinforced in their upward course by fibers derived from the quadrigeminal and geniculate bodies.

Tracing the pyramidal fibers through the medulla, they will be found to divide into two unequal portions at its lower part. The larger decussates at this point (the region of the first and second cervical nerves), constituting the decussation of the pyramids; it then crosses to the posterior part of the lateral column of the opposite side, in which it runs as the crossed pyramidal tract.

In their course these fibers give off collaterals at right angles to themselves. These pass into the gray matter, and terminate in arborizations about the root-cells of the anterior horn of the same side. The main axes end in the same manner. As these main fibers with their collaterals pass into the gray matter at various levels of the cord, the tract becomes more and more attenuated, and terminates finally in the lumbar enlargement of the cord in the neighborhood of the third or fourth sacral nerve. The smaller division of the medullary pyramids passes directly into the anterior region of the cord without decussating, and is known as the direct pyramidal tract, or the column of Türck. In its course it gives off collaterals at right angles. These pass through the anterior commissure at different levels of the cord, and end in relation with cells of the anterior horn of the opposite side. The main fibers terminate precisely in the same manner.

Thus it will be observed that the fibers of the column of Türck decussate in the anterior commissure of the cord; like the tract previously described, it becomes gradually smaller from above downward, and ends in the lower part of the dorsal cord. The axis-cylinders of the multipolar ganglion-cells of the anterior horns pass out through the anterior roots of the same side and terminate in end-plates of muscles. Déjerine, Oppenheim, Monakow, and other neurologists believe that each motor cortex sends fibers to both sides of the body, and that the decussation of the pyramids is not a complete one, a small number of the fibers running in the lateral pyramidal tract on the same side as the lesion. This is borne out clinically by the slight paresis and the plus knee-jerk on the same side, neither of which, however, approaches in degree the palsy and increased knee-jerk on the side opposite to the lesion.

Pathologic confirmation of this view has been obtained by several observers, who have found degeneration in both latero-pyramidal columns in cases of a unilateral lesion in the motor cortex.

Motor-fibers from the nuclei of cranial nerves after decussating

join with motor fibers of the internal capsule. The exact course of these fibers, however, has not been demonstrated anatomically. Since many of the muscles supplied by the cranial nerves functionate bilaterally—*e. g.* the eye-muscles and the muscles of mastication—the supposition is that in addition to fibers from its own nucleus each motor cranial nerve receives fibers from the corresponding nucleus of the opposite

side. It was Broadbent who first pointed out that parts that functionate bilaterally are supplied from both sides of the brain.

The course of the fibers of the posterior column is as follows:

The ganglion-cells on the posterior roots give rise to two fibers, fused for a short distance from the cell, but soon bifurcating. The longer of the two, the centrifugal fiber, extends to the surface and terminates in pointed or bulbous endings in the epidermis, or in special sensory nerve-endings in tactile cells, tactile corpuscles, or end-bulbs. The centripetal fibers or axons penetrate the cord, and divide in the white matter into ascending and descending fibers. The former may be either long or short. The short fibers are vertical at first, but finally bend into the gray matter, and end in relation with certain cells of the anterior cornua, forming perhaps a part of the reflex arc. Their collaterals end in a similar manner.

The long fibers extend up the

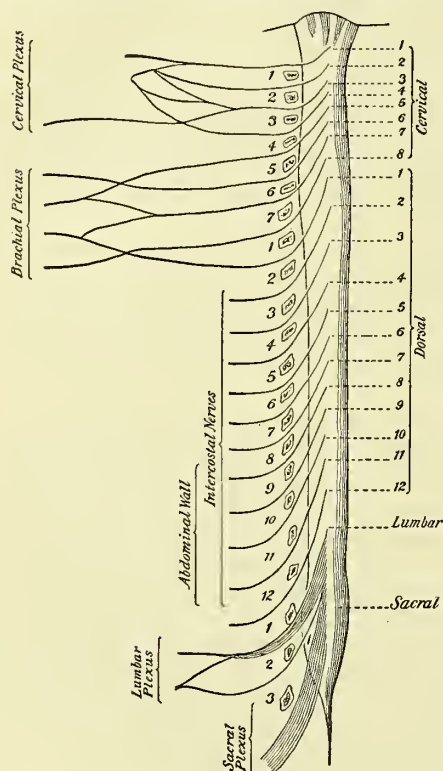


FIG. 72.—Diagram showing the groupings and plexuses of the spinal nerves (redrawn after Baker).

cord to the medulla, ending in the usual manner in the gray nuclei of the columns of Goll and Burdach; these are known as the *nucleus gracilis* and *nucleus cuneatus*, respectively. They also give off collaterals in their course. The descending fibers, on the other hand, are all short, and probably constitute the so-called *comma tract* of Schultze.

Since fibers continue to enter the cord at different levels, those that have entered below are pushed more and more toward the median line. It will thus be seen that the column of Goll is made up almost entirely of long fibers, and that the column of Burdach also contains long fibers, although it is probable that the short ones predominate. The long fibers are concerned in muscular coördination and equilibrium. It is likely that the fibers of pain and temperature sense, although entering by the posterior roots, do not pass up through the posterior columns, but rather through the gray substance of the spinal cord.



Since the post-natal growth of the vertebræ is more rapid than that of the cord, it follows that the spinal nerves assume a more and more oblique position, until finally the spinal segments, each of which consists of an anterior and posterior nerve-bundle with a transverse plane of white substance, lie considerably above the vertebræ after which they are named (see Fig. 67). The following table (Starr, modified by Mills and Dana from the experimental and clinical studies of Thorburn and others) shows the localization of function (not organs) in the different segments of the cord:

*Localization of the Functions of the Segments of the Spinal Cord.*

SEGMENT.	MUSCLES.	REFLEX AND CENTERS.	SENSATION.
First cervical.	Rectus laterales. Rectus capitis. Anticus and posticus. Sterno-hyoid. Sterno-thyroid.		
Second and third cervical.	Sterno-mastoid. Trapezius. Scaleni and neck. Omo-hyoid. Diaphragm.	<i>Hypochondrium</i> (?). Sudden inspiration produced by sudden pressure beneath the lower border of the ribs.	Back of head to vertex and neck. (Occipitalis major, occipitalis minor, auricularis magnus, superficialis colli, and supraclavicular.)
Fourth cervical.	Diaphragm. Deltoid. Biceps. Coraco-brachialis. Supinator longus. Rhomboid. Supra- and infra-spiratus.	<i>Pupillary</i> (fourth cervical to second dorsal). Dilatation of the pupil produced by irritation of the neck.	Neck. Shoulder, anterior surface. Outer arm. (Supraclavicular, circumflex, external musculo-cutaneous, cutaneous.)
Fifth cervical.	Deltoid. Biceps. Coraco-brachialis. Brachialis anticus. Supinator longus. Supinator brevis. Deep muscles of shoulder-blade. Rhomboid. Teres minor. Pectoralis (clavicular part). Serratus magnus.	<i>Scapular</i> (fifth cervical to first dorsal). Irritation of skin over the scapula produces contraction of the scapular muscles. <i>Supinator longus</i> . Tapping the tendon of the supinator longus produces flexion of forearm.	Back of shoulder and arm. Outer side of arm and forearm to the wrist. (Supraclavicular, circumflex, external cutaneous, internal cutaneous, posterior spinal branches.)
Sixth cervical.	Deltoid. Biceps. Brachialis anticus. Subscapular. Pectoralis (clavicular part). Serratus magnus. Triceps. Pronators. Rhomboid. Latissimus dorsi.	<i>Triceps</i> (fifth to sixth cervical). Tapping elbow tendon produces extension of forearm. <i>Posterior wrist</i> (sixth to eighth cervical). Tapping tendons causes extension of the hand.	Outer side and front of forearm. Back of hand, radial distribution. (Chiefly external cutaneous, internal cutaneous, radial.)
Seventh cervical.	Triceps (long head). Extensors of wrist and fingers. Pronators of wrist. Flexors of wrist. Subscapular. Pectoralis (costal part). Serratus magnus. Latissimus dorsi. Teres major.	<i>Anterior wrist</i> (seventh to eighth cervical). Tapping anterior tendons causes flexion of wrist. <i>Palmar</i> (seventh cervical to first dorsal). Stroking the palm causes closure of the fingers.	Radial distribution in the hand. Median distribution in the palm, thumb, index, and one half of the middle finger. (External cutaneous, internal cutaneous, radial, median, posterior spinal branches.)
Eighth cervical.	Triceps (long head). Flexors of wrist and fingers. Intrinsic hand-muscles.	.....	Ulnar area of hand, back, and palm, inner border of forearm. (Internal cutaneous, ulnar.)
First dorsal.	Extensors of thumb. Intrinsic hand-muscles. Thenar and hypothenar muscles.	.....	Chiefly inner side of forearm and arm to near the axilla. (Chiefly internal cutaneous and nerve of Wrisberg or lesser internal cutaneous.)
Second dorsal.	.....	.....	Inner side of arm near to and in the axilla. (Intercosto-numeral.)

SEGMENT.	MUSCLES.	REFLEX AND CENTERS.	SENSATION.
Second to twelfth dorsal.	Muscles of back and abdomen. Erectores spinæ.	<i>Epigastric</i> (fourth to seventh dorsal). Tickling mammary region causes retraction of the epigastrium.  <i>Abdominal</i> (seventh to eleventh dorsal). Stroking side of abdomen causes retraction of belly. Vaso-motor centers. Second dorsal to second lumbar.	Skin of the chest and abdomen, in bands running around and downward, corresponding to spinal nerves. Upper gluteal region. (Intercostals and dorsal posterior nerves.)
First lumbar.	None.	<i>Cremasteric</i> (first to third lumbar). Stroking inner thigh causes retraction of scrotum.	Skin over groin and front of scrotum. (Iliohypogastric, ilioinguinal.)
Second lumbar.	Vastus internus.	<i>Patellar</i> . Striking patellar tendon causes extension of the leg.	Outer side and upper front of thigh. Lumbar region. (Genitocrural, external cutaneous.)
Third lumbar.	Sartorius; adductors of thigh.		Front and outer side of thigh. Inner side of leg and foot.
Fourth lumbar.	Flexors of thigh. Extensors of knee. Abductors of thigh.	<i>Gluteal</i> (fourth to fifth lumbar). Stroking buttock causes dimpling in fold of buttock.	Inner side of thigh, leg, and foot. (Internal cutaneous, long saphenous, obturator.)
Fifth lumbar.	Outward rotators. Flexors of knee. Flexors of ankle. Peronei. Extensors of toes.	<i>Achilles tendon</i> . Overextension causes rapid flexion of ankle, called <i>ankle-clonus</i> .	Back of thigh and outer side of leg and ankle; sole; dorsum of foot. (External popliteal, external saphenous, musculocutaneous, plantar.)
First and second sacral.	Calf-muscles. Glutei. Peronei. Extensors of ankle. Small muscles of foot.	<i>Plantar</i> (fifth lumbar to second sacral). Tickling sole of foot causes flexion of toes and retraction of leg.	Back of buttock and thigh, side of leg and ankle; sole; dorsum of foot.
Third, fourth, and fifth sacral.	Perineal. Muscles of bladder, rectum, and external genitals.	Genital center. Vesical center. Anal center.	Circumanal region, anus, rectum, penis, urethra, vagina, perineum. (Small sciatic, pudic, inferior hemorrhoidal, inferior pudendal.)

To the foregoing table, which illustrates spinal localization, should be added another, showing what functions reside in the pons and medulla, as follows:

## NUCLEI.

- III. { Sphincter iris. Ciliary muscles.  
Levator palpebræ superioris. Rectus internus (in convergence).  
Rectus superior. Rectus inferior.  
Obliquus inferior.
- IV. { Obliquus superior.  
(Upper facial group.)

- V. { (Associated movement of levator palpebræ.)  
Muscles of lower jaw.

- VI. { Rectus externus. Rectus  
inter. of opposite side  
in lateral movements.

## VII.—Facial muscles.

- XII. { (Lower facial group.)  
Muscles of tongue.

- IX. { Muscles of pharynx.  
X. { Muscles of esophagus.  
XI. { Muscles of larynx.

*Sensory Cortical Area.*—Owing to the extensive compensation of sensory fibers, by means of which each side of the brain sends fibers to both sides of the body, it is impossible to map out the center with precision.

It is generally believed, for reasons already stated, that the central convolutions (motor area) contain muscular and tactile sensory functions. These are also spread out over the parietal lobe, and it is possible, indeed probable, that the sensory zone extends to the mesial surface of the hemisphere, as does the motor area. That this is the chief sensory center, as claimed by some observers, is, however, very questionable.

From the cuneus, fibers pass to the pulvinar, forming an optic radiation of the Gratiolet. From the pulvinar they apparently pass to the external geniculate bodies, and thence to the anterior corpus quadrigeminum. The optic tracts arise by two roots that curve round the crista on either side and unite immediately in front of the tuber cinereum. Fibers from the two tracts pass to the homologous sides of both retinae; therefore the lesions posterior to the chiasm give rise to blindness of half of the retina on the same side.

*Visual Centers.*—The exact center for ordinary vision is in the cortex of the occipital lobe of the inner surface in the region of the calcarine fissure. A higher center exists, probably located in the angular gyrus, and a lesion of which produces mind-blindness: this is a condition in which vision is not lost, but the seen objects are not recognized by the individual. Ferrier says that a lesion in this region sometimes gives rise to crossed amblyopia. The eye opposite to the lesions is chiefly affected, though vision is also restricted in the eye on the same side of the lesion (visual tract).

*Olfactory Center.*—This is located in the anterior part of the uncinate convolution, on the inner surface of the temporal lobe. It is possible, too, that fibers pass from this region through the anterior commissure to the cortex of the opposite hemisphere.

*Auditory Center.*—A lesion in the posterior part of the first temporal convolution produces a deafness in the opposite ear that is transient in character, owing to compensation. Bilateral lesions produce complete deafness. Mind-deafness, or an inability to understand spoken words, has resulted from a lesion in the first temporal convolution of the left side.

*Speech Center.*—The articulate speech center is located in the posterior part of the left third or inferior frontal convolution, and in the adjacent part of the ascending frontal in right-handed people (but on the right side in left-handed persons).

It is not known exactly what part the island of Reil plays in articulate speech. Word-blindness results from a lesion in the angular gyrus. Word-deafness results from a lesion in the posterior part of the first left temporal convolution.

*Taste Center.*—The area of cortical representation is unknown. By some it is located in the gyrus hippocampus.

*Psychic Centers.*—It is possible that the frontal lobes, anterior to the precentral fissure, contain the psychic centers. Such extensive compensation probably exists that no ordinary lesion produces mental aberration, but these centers are probably represented by the whole cortex.

The function of the cerebellum is that of coördination. Fibers pass from its cortex to that of the cerebrum, and *vice versa*. The impressions derived from the cerebrum are believed to be inhibitory.

Peripheral impressions reach the cerebellum through the direct cere-



bellar tracts of the lateral columns of the cord, and also from fibers derived from cells in the nuclei of the columns of Goll and Burdach.

Motor impulses run from the cerebellar cortex to the motor region of the cerebral cortex by way of the superior or middle peduncle, and by way of the inferior peduncle (restiform body) to the multipolar ganglion-cells of the anterior horns.

#### GENERAL AND TOPICAL DIAGNOSIS.

Nervous diseases are usually spoken of either as being *functional* or *organic*; but, as our methods of research become more refined and our technic more perfect, the breach between these two groups is being gradually but steadily lessened.

Granting this, they all really become organic diseases, though some in which neither macroscopic nor microscopic change has ever been discovered are called functional for the sake of convenience.

**Organic nervous diseases** may be produced by two types of lesions:

1. *Irritative*, causing an increase of function, continuous or intermittent.
2. *Destructive*, resulting in paralysis of motion or sensation, or both.

Irritative lesions are prone to become destructive in course of time. They may be operative in the upper segment, which includes the brain and fibers leading to or from it as far as the ganglion-cells of the cord; or in the lower segment, including the multipolar ganglion-cells of the anterior horn, together with the peripheral motor and sensory nerves.

When a complete pathway is involved a systemic disease is said to be produced. When two or more paths or neuron complexes are simultaneously involved combined systemic disease results.

Brain-lesions may be (a) focal or (b) diffuse. Cord-lesions are either (a) transverse, (b) focal, or (c) insular (a series of foci).

Cord-lesions result in ascending or descending degeneration, the destructive process travelling, as a rule, in the direction in which impulses are normally transmitted. In the fillet degeneration may extend up or down.

The theory has been advanced that the vulnerability of the tracts of the spinal axis is in direct proportion to the degree of their functional activity; hence the reflex (sensory and pyramidal) tracts are more likely to degenerate under nutritional disturbances or toxic processes than other parts.

It has been supposed that the tardy myelination of the pyramidal tracts predisposes to various nervous maladies, and particularly to those of a convulsive type. The following may be accepted as a general rule: the motor-nervous system is the last to develop, the first to lose, and the last to regain, its function; while the sensory nervous system is the first to develop, the last to lose, and the first to regain, its function. In making a diagnosis it is, therefore, of the utmost importance to try to determine the locality and extent of the morbid process, and to ascertain whether the lesion is a focal or systemic one. The symptomatology of systemic diseases is pretty constant, and, except in their very incipency, they are usually not difficult of diagnosis. The symptoms of focal diseases, on the other hand, vary, of necessity, according to the location

of the focus. They are often difficult and at times impossible to diagnose. Especially is this true of lesions occurring in the frontal lobes of the cerebrum, in the basal ganglia, and in the cerebellum.

Since the study of the motor centers and tracts has been pursued with so much more success than that of the sensory system, positive or negative motor phenomena occurring in the course of nervous diseases furnish us with much more valuable information than do sensory manifestations.

Further, motor symptoms are objective, and consequently appeal to us in a much greater degree than the sensory symptoms, which are purely subjective, and the elicitation of which depends so much upon the mental capability of the patient.

Irritative motor-lesions produce, according to the degree of irritation, either fibrillary muscular twitchings or mild or severe convulsions, tonic or clonic in character.

Destructive motor-lesions, according to their extent, produce mere muscular weakness, paresis, or actual paralysis of a single muscle, groups of muscles, or of the entire musculature of one or more limbs.

Irritative sensory lesions give rise to neuralgia, hyperesthesia, or hyperalgesia.

Destructive sensory lesions cause a more or less complete absence of sensation, as analgesia, anesthesia, or loss of temperature-sense.

*Upper-segment or Upper-system Diseases.*—A lesion occurring in the motor pathway anywhere between the cortex and the multipolar cells of the anterior horns (but not including the latter) gives rise to the following symptom-complex: Loss of motion, both automatic and volitional, and chiefly on the side of the body opposite to the lesion. The paralysis is usually spastic in type. The muscles resist passive movements, showing that their tone is increased. This is relative, and is due to the removal of cerebral inhibition, which allows the lower centers free play. They also tend to undergo shortening, and contractures result. Reflexes are increased chiefly on the side opposite the lesion, but also on the same side, the increase being the result of the removal of cerebral influences.

Owing to inactivity, the muscles of the paralyzed members undergo a more or less marked atrophy, though there are no degenerative changes, since the neuron bodies are intact. For the same reason the response to electric stimulation is not interfered with.

An irritative lesion of this upper system, particularly when operative in or upon the cortical region, gives rise to tonic or clonic convulsive movements. When the lesion is localized to a single center, focal or so-called Jacksonian epilepsy results. The cortex is wonderfully tolerant, when the lesion is of gradual onset and the parts accommodate themselves to the slowly increasing pressure. However, a local irritative lesion may at first cause widespread symptoms, due, as Nothnagel pointed out, to pressure, vascular disturbances, or irritative inhibition.

*Lower-segment or Lower-system Diseases.*—This includes the peripheral neuron system. Since there is no crossing of the fibers, the lesion and resulting paralysis are on the same side of the body. The paralysis, however, is of the flaccid, flail-like variety, hypotonus being present. The muscles offer no resistance whatever to passive movement, contrac-

tures do not occur, and reflexes are lost. Extreme degrees of wasting occur in this type of paralysis, owing partly to disuse, but chiefly to the fact that the neuron body, the nutritional or trophic center for the fiber, is injured. Pathologic changes therefore take place in the muscles themselves, and form a true degenerative atrophy. The protoplasm first becomes granular, and then fatty; it then breaks down and is absorbed. Its place is taken by the connective tissue, which is both relatively and absolutely increased, so that in the course of time fibrous masses alone remain. Electric changes also occur. The muscles first cease to respond to the faradic current, and soon respond in an abnormal manner to the galvanic. Instead of short, sharp contractions, they react in a slow, wavy manner, ACC being stronger than KCC. Irritative lesions of this system cause fibrillary muscular contractions and peripheral convulsions, of which laryngismus stridulus is a type.

## I. DISEASES OF THE PERIPHERAL NERVES.

### NEURITIS.

**Definition.**—An inflammation of a nerve or of its fibrous envelope.

**Pathology.**—A true neuritis is almost always an inflammation of the nerve-sheath or of the septa between the fasciculi, and usually begins as a perineuritis. The so-called parenchymatous neuritis is really a degenerative process; it is prone to follow neuritis, and is the result of excessive or prolonged irritation or of pressure by the products of inflammation. The affected nerve becomes red and swollen. The sheath becomes hyperemic and the seat of a round-cell infiltration.

We may have a *perineuritis* or an *interstitial neuritis*. Again, these may be *focal* or *diffuse* (*disseminated*), involving limited patches or continuous areas of a nerve. Finally, many nerves may be simultaneously affected, constituting a *multiple neuritis*. In the parenchymatous form the ordinary signs of inflammation are absent. The nuclei of the sheath increase in size and number, and the protoplasm about them increases in amount. The white substance of Schwann becomes segmented, breaks up into droplets, then becomes granular and fatty, and is finally absorbed. The axis-cylinder becomes discontinuous at the site of disorganization of the myelin. Ultimately, there may be seen scattered promiscuously among the more or less healthy fibers the withered nerve-sheaths, containing many nuclei, some granular debris, and pigment. Occasionally fatty aggregations occur along the nerve. Leyden has termed this condition *lipomatous neuritis*, but it is not worthy of a special name, as it is only a stage in the ordinary degenerative process.

Parenchymatous degeneration is similar to the secondary or Wallerian degeneration previously mentioned. It is the chief lesion in multiple neuritis, though in this disease changes have also been found in the multipolar ganglion-cells of the anterior horns.

**Etiology.**—(a) *Focal neuritis* may be due to—(1) Exposure or cold (the so-called *rheumatic neuritis*). (2) Extension of inflammation from



neighboring parts. (3) Traumatism—wounds, compression, excessive stretching resulting from fractures or dislocation. (4) Microbic and autogenetic poisons.

(b) *Multiple neuritis* may be due to—(1) Poisons of extrinsic origin—alcohol, carbon bisulfid, lead, arsenic, mercury, ether. (2) Poisons resulting from the infectious fevers (typhoid, diphtheria, variola, typhus, leprosy, beri-beri, measles, syphilis, tuberculosis, septicemia, malaria, influenza). (3) Cachexias, anemia, carcinoma. (4) Auto-intoxication. (5) Cases arise in which no definite cause can be ascertained; these are the so-called *idiopathic* or spontaneous cases.

**Symptoms.**—(a) *Focal Neuritis*.—In localized neuritis the symptoms vary according to the function of the nerve involved. In the case of a sensory nerve there is pain, usually of a boring or shooting character, along its course and distribution. There is also tenderness on pressure along the nerve, and especially at its point of emergence from bony canals. Weir Mitchell believes this to be due to irritation of the *nervi nervorum*. The skin is generally hyperalgesic (though tactile sensation is often lowered), reddened, sometimes edematous, and local sweatings may occur. In the more chronic cases trophic symptoms eventually arise, as glossiness of the skin and an impaired growth of the nails. When a motor nerve bears the brunt of the attack fibrillary twitchings will be observed in the muscles it supplies, and are soon followed by more or less impairment of motion, even amounting to paralysis; sometimes contractures occur, and ultimately wasting of the muscles, and even reactions of degeneration, take place. When both motor and sensory nerves are simultaneously involved the symptoms will necessarily partake of a mixed character. The constitutional symptoms are, as a rule, of little moment.

(b) *Multiple neuritis* is an involvement of the peripheral nerves in various parts of the body, affected simultaneously or in quick succession, and due to endogenous or exogenous poisons. Lettsom's paper, published in 1789, embodied the first description of the disease.

Among cases due to poisons of extrinsic origin is *alcoholic neuritis*. In 1822, James Jackson of Boston clearly gave its clinical history, though Dumesnil in 1864 was the first to publish the result of an autopsy upon a case. Other pioneers were Leyden, Buzzard, and Ross. This is the most common type of multiple neuritis, and occurs oftener among women than men. It results from spirit-drinking in moderate amounts and continued over a long time. The onset is generally slow, being preceded by gastric catarrh, insomnia, tingling of the extremities, a rapid, weak heart, and a tendency to sweating on exertion. Some muscular twitching and paresis may exist contemporaneously, but the loss of power soon becomes more marked—first in the lower and then in the upper extremities, the extensors being chiefly affected. Wrist-drop and foot-drop follow. Occasionally paraplegia and, more rarely still, a loss of control of the bladder and rectum take place. Fever is rarely present. Sensory symptoms may vary from the tingling or numbness already noted to burning or boring pains of great severity. The skin is hyperesthetic at first, at all events. Later, paresthesiæ develop, with anesthesia and a more or less decided loss of muscular sense. The muscles are tender when touched.

The cutaneous reflexes are preserved unless the anesthesia is marked. The knee-jerks are generally lost, though exceptionally they may be increased. In the less severe cases a certain amount of incoördination may be present. When this is the case the absence of the knee-jerk, the loss of muscular sense, ataxia, and the pains in the extremities simulate locomotor ataxia, and the term *pseudo-tabes* has been applied to the condition. Vaso-motor and trophic symptoms appear, and in some cases the special senses are involved (impairment of vision, amblyopia, limitation of the color-field). The cerebral symptoms are important. They may be so slight as to consist merely of loss of memory, irritability, perhaps an hallucination or illusion (particularly after night-fall, and especially if the patient has had insomnia), or they may be of the type and degree seen in general paralysis. The duration of an attack varies from a few weeks to a year or so.

*Arsenic neuritis* differs from the above in that the head-symptoms are generally absent. The onset may be much more abrupt and the course is usually shorter.

*Carbon bisulphid neuritis* occurs chiefly in workers in rubber-factories. There are noted intense frontal headache, giddiness, marked excitability, muscular cramps, and possibly convulsions. *Saturnine neuritis* is confined to motor nerves, and especially to those of the upper extremities. Very rarely some disturbance of sensibility may result. Lesions of the anterior cornua are more likely to occur in saturnine multiple neuritis than in any of the other varieties. Head-symptoms are not common, but optic neuritis and convulsions may occur.

Cases due to an attack of some infectious disease may be local or multiple, and generally present the same symptoms as neuritis due to any other cause. (1) *Malarial Neuritis*.—According to Romberg, malaria gives rise at times to "intermittent paraplegia." The legs of the patient suddenly become paralyzed, with or without alteration of sensation or loss of control of the sphincter. That the cause is probably malarial is shown by the fact that the condition is periodic, each attack subsiding with a critical sweat, and finally yielding to quinin. (2) *Recurring Multiple Neuritis*.—A few cases have been reported in which attacks of more or less widespread paralysis, due to neuritis, have recurred.

*Spontaneous* or the so-called *idiopathic neuritis* does not differ from the general type of the disease, except that no cause can be discovered to account for it.

**Diagnosis.**—This does not present any difficulty, as a rule. The spontaneous cases, in the early stages, may simulate *acute spinal paralysis* or *acute ascending paralysis*. The fever, palsy, electric change, and the loss of knee-jerks are common to both, but in *acute spinal paralysis* there are never any sensory symptoms. The palsy in idiopathic cases rapidly spreads, but soon subsides again.

In other forms of peripheral neuritis the onset is not only apt to be less abrupt, but some sensory symptoms are almost invariably present; the distribution of the palsy is more symmetric bilaterally, and after it has reached its acme no improvement takes place for a few weeks or months. In *ascending paralysis* there are no sensory symptoms, the knee-jerks are preserved, there is neither muscular atrophy nor electric

change, and the order in which the paralysis supervenes differs from that of peripheral neuritis.

Cases of pseudo-tubes are sometimes confounded with *locomotor ataxia*. The main points of differentiation are included in the following table:

PSEUDO-TUBES.	LOCOMOTOR ATAXIA.
The course is shorter, and often results in recovery.	The course is progressive from bad to worse, and chronic in nature.
Pain is never of the fulgurant type.	Fulgurant pains often are present. Pain-crises are almost diagnostic.
There is tenderness over the nerve-trunks.	There is no tenderness over the nerves.
Sensory disturbances are more marked (tingling and numbness).	Sensory disturbances are less marked.
Argyll-Robertson pupil is absent.	Argyll-Robertson pupil is present.
There is a "foot-drop," with the typical "steppage" gait.	No "foot-drop." The toes are raised, and the foot is brought down flatly, with the heel first.
Paralysis is often present.	There is no actual loss of power.

**Prognosis.**—Peripheral neuritis may terminate in one of the following ways, according to Drs. Gibson and Fleming<sup>1</sup>: 1. In complete recovery; 2. With damaged peripheral nerves; 3. With injury to the central nervous system, such as to cause symptoms of ataxia, spastic paraplegia, or disseminated sclerosis; 4. In death, from failure of the organic centers, especially that of respiration. The prognosis is generally good, though in the acute variety (from any cause) it should be guarded, and occasionally is grave. Exposure and chill, alcohol, diphtheria, and beri-beri give rise to the most serious types, and often cause death by failure of the heart or respiration or by coagula in the vessels. Mild cases may entirely recover in a few weeks, while severe ones often require a year or two.

**Treatment.**—First ascertain the cause, and, if possible, remove it. It may be unwise in alcoholic cases to stop the alcohol suddenly, but each case must be judged on its merits. Rest is very important, and all sources of worry should be stopped. Locally, anodynes may be employed and the part wrapped in cotton wool. In febrile cases, especially in the earlier stages, the salicylates are valuable. The general health should be toned up by strychnin and tonics, and by nourishing but easily digestible food. Further medication will depend upon the etiology, quinin being demanded in malarial, and mercury or the iodids in syphilitic cases. As soon as the acute symptoms have subsided massage and passive movements should be begun, galvanism applied to the muscles, and warm-water or sulphur baths administered.

## BERI-BERI.

THIS is a tropical disease, characterized by weakness, wasting of the muscles, paralysis, anasarca, anemia, numbness, pain, areas of anesthesia, and diminution or loss of tendon-reflexes. There are two forms, the *acute* and the *chronic*. Its etiology is obscure, though it is apt to supervene upon any condition that impoverishes the physical or nervous

<sup>1</sup> *Edinburgh Hospital Reports*, vol. iii.



vitality. Intestinal parasites have been said to cause it. Ogata of Tokio has described a specific bacillus; Pikelharing and Winkler, however, claim that it is due to a micrococcus. Whatever its cause, the researches of these latter observers, together with those of Baelz and Sheube in Japan, prove it to be a peripheral multiple neuritis. The symptoms of the *acute form* are fever, anemia, general edema, effusion into the serous cavities, dyspnea, precordial pain, vomiting, and peripheral paralysis. Death often results, even in a few days, from emboli or thrombi in the pulmonary or systemic circulation. In the *chronic form* the symptoms are less pronounced. The face is apt to be puffy, and palpitation and serious cardiac dilatation may occur. The gait is tottering, the muscles are somewhat wasted, the tendon-reflexes are lost, and paræsthesiæ develop.

The cases associated with the cachectic states may be general; though usually they are local and of the interstitial variety of neuritis.

The cases due to auto-intoxication are usually associated with fever, and at first simulate rheumatism or some infectious disease. Soon, however, the tingling, pain, palsy, loss of the knee-jerks, and anesthesia reveal the neuritis. Death may result from cardiac or respiratory paralysis. When life is spared the convalescence is exceedingly slow.

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## NEUROMATA.

NEUROMATA, or tumors of nerves, have been described as (a) true and (b) false.

(a) *True neuromata* consist of medullated or non-medullated nerve-fibers (the myelinic and amyelinic varieties—Virchow), and rarely of ganglion-cells also.

(b) *False neuromata* contain no nerve-elements. The growth is situated on the nerve-trunk itself, and consists of either fibrous, myxomatous, gliomatous, or sarcomatous tissue.

Neuromata have also been classified according to their situation as (1) Stump neuromata, or bulbous nerves; (2) Subcutaneous neuromata, or *tubercula dolorosa*; (3) Nerve-trunk neuromata; (4) Plexiform neuromata.

(1) *Stump neuromata* develop on stumps or on the ends of divided nerves as the result of traumatism. They may consist of fibrous tissue, but are usually myelinic.

(2) *Subcutaneous tumors*, or *tubercula dolorosa*, are painful, as the latter word implies, and are apt to be multiple. In individuals so afflicted nerve-trunk neuromata may coexist.

(3) *Nerve-trunk neuromata* are usually multiple. In one case quoted by Gowers as many as 3020 were found. They may be true or false. In the former case the nerve-fibers are less apt to be interfered with than in the heterologous growth.

(4) *Plexiform neuromata* consist of beaded and tortuous, interlacing neural cords. They are usually congenital.

**Etiology.**—Neuromata may be due to traumatism. When multiple,

however, they are usually hereditary, occurring in families of a neurotic or strumous diathesis. They are most commonly found in men.

**Symptoms.**—There may be none. When present their character necessarily depends on the nature of the nerve involved and whether the lesion is an irritative or destructive one. More or less pain, numbness or tingling, paraesthesia, and palsy are among the most common symptoms. Various reflex manifestations have been described, and epileptiform convulsions have been attributed to their presence.

**Treatment.**—Apart from anodynes, operative measures are alone of value, except when the tumors are the result of syphilis, as occasionally happens; in such cases specific treatment must be employed.

It must not be forgotten, however, that stump neuromata may occur in those hereditarily predisposed, in which case, as Bowlby has pointed out, their removal will almost surely be followed by a return.

## NEURALGIA.

**Definition.**—Neuralgia (*nerve-pain*) is the result of some irritation directly or indirectly applied to a nerve. While this is true of all pain, yet the special nerve-pain under consideration presents the following characteristics: 1st. In its distribution it follows the course of a nerve-trunk or its branches. 2d. It shows a tendency to shift from place to place. 3d. The presence of painful points (*points douloureux*). 4th. Intermission and remission of pain.

The pain of neuralgia varies both as to character and intensity. It may be merely a mild ache, or, on the other hand, it may give rise to the most excruciating agony; it may be of a throbbing, boring, tearing, shooting, or burning character, or it may come on in shock-like paroxysms. Any nerve in the body may be affected. Quite often one can find no definite cause of the neuralgia, and as we are not certain as to its ultimate pathology, it may be due to some slight inflammation of the nerve, or to hyperemia, ischemia, exudation, and the like.

Among the *predisposing causes* are—(1) Age, the condition being most common in those between thirty-five and fifty years, and less so above that age. It is least common in children.

(2) Sex. On the whole, neuralgia is most common in women, though the severer grades are found quite as frequently in men. Sciatica is more common in men, while trigeminal neuralgia occurs more frequently in women.

(3) Heredity. Neuralgia is very prone to occur in a family in which hysteria, epilepsy, or other neurosis or psychosis is present.

(4) The general physical condition. In persons reduced by illness or by mental or physical exertion, and in anemia, neuralgia is common.

(5) Occupation. Painters and workers among metallic dust are specially predisposed.

The *exciting causes* are—(1) Exposure to cold and wet. (2) Mechanical, chemical, or thermal irritation, including compression. (3) Traumatism. (4) Neuromata. (5) Infectious diseases. (6) Rheuma-

tism and gout. (7) Endogenous or exogenous poisons. (8) Local disease in the course of the nerve, or of the mouth, nose, or orbit.

We are hardly justified to-day in speaking of idiopathic neuralgia. Such cases probably result from some endogenous or exogenous poison, an auto-intoxication, or malaria, rheumatism, syphilis, some metallic poison, or alcohol or tobacco. Finally, a form exists which we speak of in no certain manner as "reflex neuralgia," and which is said to result from disease of the sexual or other organs often remote from the painful nerves.

**General Symptomatology.**—Neuralgia may be of sudden or slow onset, with or without prodromata. When the latter exist they consist of a sense of uneasiness, perverted sensations, chilliness, and stinging or slight burning pains. The pain may be of the character previously described, either strictly localized or radiating to neighboring nerves, and may be aggravated by drafts, movements, or mental perturbation. On pressure certain tender or painful spots will be found, especially where the nerves emerge from deeper parts and become superficial. The affected part is usually hyperesthetic; occasionally, however, it is anesthetic, and may continue so for some time after an attack.

Reflex muscular contraction may be present in proportion to the intensity of sensory irritation. Vaso-motor symptoms manifest themselves in the flushing or blanching of the affected part and in increased secretions, as sweating.

*Trophic disturbances* may result in temporary or permanent changes. To the former belong the herpetic and urticarial eruptions, while the latter groups include change of color in, loss of, or overgrowth of the hair, various changes in the skin (as pigmentation and morphea, and even ulceration, though in the latter instance there is probably a more profound pathologic change than that which we regard as the cause of neuralgia). Unless the attacks are severe or prolonged, however, the general system seldom suffers.

Neuralgia may be divided into the following groups: neuralgia of the head, neck, trunk, upper and lower extremities; neuralgia of the genitals and rectal region; and visceral neuralgias.

#### NEURALGIA OF THE HEAD.

Trigeminal neuralgia (*Tic douloureux*) may manifest itself in any one or all of the branches of the fifth nerve.

**Neuralgia of the First Branch** (*Ophthalmic Neuralgia*).—The chief points of pain in this variety are the supra-orbital foramen (the exit of the nerve), the nose, the eyeball, and parts around the eye.

**Supra-maxillary neuralgia** (involving the second branch of the fifth, the supra-maxillary nerve). The tender points here are the infra-orbital foramen (the exit of the nerve), the cheek, side and cavities of the nose, the upper lip and gums, and the zygoma.

The **infra-maxillary division**, the third branch of the fifth. The mental foramen is the point of greatest tenderness; other spots, however, are the temporal region, and along the tongue and lower lip.



## NEURALGIA OF THE NECK AND TRUNK.

The cervical branches of the dorsal and lumbar nerves are involved in this group.

1. *Cervico-occipital neuralgia*, occurring in the occipital and posterior parietal region, is apt to be quite severe, but when not due to spondylitis (the result of caries) or neoplasms the prognosis is fair. It is sometimes the result of direct pressure, as in carrying heavy loads on the neck and shoulders. The painful spot is found between the mastoid process and upper cervical vertebræ. Falling of the hair may also occur. This is much more apt to take place, however, when the occipitalis minor is involved, as it is said that the latter is generally a syphilitic neuralgia.

2. *Phrenic neuralgia* has been described, but is a rare condition. The pain is in the lower anterior thoracic region, at the points of insertion of the diaphragm.

3. *Intercostal Neuralgia*.—The middle intercostal nerves are most liable to be affected, and generally on the left side. The posterior dorsal branches are seldom involved. When specially severe and persistent, intercostal neuralgia may be a symptom of disease of the cord or its membranes, aneurysm of the aorta, neoplasms, or disease of the vertebræ or ribs. Traumatism and cold also give rise to it. This form of neuralgia is most common in women, the painful spots being at the extremity and at the middle of the ribs. The pain is of a sharp, lancinating character and radiates along the nerve. It is intensified by all movements of the chest; hence the affected side is more or less fixed. Herpes may develop, but in such cases it is probable that a true neuritis exists.

4. *Mastodynia* is really a variety of intercostal neuralgia, and occurs almost solely among women. It is very painful, and gives rise to the development of tender "lumps" in the breast, simulating malignant disease. The paroxysms are often accompanied by vomiting.

5. *Lumbo-abdominal neuralgia* is not a common form. The pain is chiefly in the lumbar region, though the hypogastrium, genitals, and buttocks may also be involved.

## NEURALGIA OF THE EXTREMITIES.

*Cervico-brachial neuralgia* occurs in the distribution of the four lower cervical nerves. When the condition is bilateral we should look for disease of the cord or membranes, for new growths, or for disease of the vertebræ. When unilateral, any of the causes already enumerated may be operative. The radial and ulnar nerves are more frequently affected than the median. The pain is most apt to be distributed along the whole course of the nerve, but painful points are found in the following situations—in the axilla; over the brachial plexus; on the shoulder, where the cutaneous branches of the circumflex nerve emerge through the deltoid muscle; about the middle of the outer surface of the upper arm; over the ulnar nerve; in the sulcus between the olecranon and epitrochlea; also near the wrist and at the bend of the elbow over the musculo-spiral nerve.

*Femoral or crural neuralgia* is a somewhat rare type that attacks the anterior surface of the thigh, the knee-joint, and the inner surface of the leg and foot.

*Obturator neuralgia* is distributed along the inner side of the thigh down to, and including, the knee-joint. This form is common in women subject to ovarian diseases.

*Sciatica* stands next to trigeminal neuralgia in the order of frequency, and is by far more common in men than women. In addition to the ordinary causes of neuralgia—exposure, compression, traumatism—the condition may be an early symptom of tabes or it may be due to constipation or hemorrhoids. The painful points are in the gluteal region and the popliteal space or malleolar region, though tenderness may be elicited along the whole course of the nerve. The pain is sharp and shooting, or more often of a tearing variety. Fine or coarse tremors or spasms may be present, together with some disturbance of sensation and loss of power. Herpes occasionally develops along the course of the nerve. This form of neuralgia is quite common, and is generally easy to recognize, but it may be simulated by hip-joint disease, psoas abscess, or lumbago.

Sciatica may also be caused by neuritis, the result of exposure or compression by pelvic growths, or by the fetal head during labor. In such cases there is, as a rule, slight fever, and the tenderness on pressure and the degree of pain are infinitely more severe than neuralgia. In severe cases the patient is bed-ridden, but in milder attacks he can be about; walking greatly increases the pain, however. It is slightly mitigated by relieving the tension on the nerve, by bending the knee, and walking on the toes. It is an obstinate condition, and relapses are common.

The *diagnosis* is generally not difficult. The distribution of the pain, the location of tender points, and the character of the gait suffice to prevent error, as a rule. A rectal or vaginal examination should be made to determine whether it is a primary disease or secondary to some pelvic condition, for clearly, if the cause can be removed, the prognosis will be good.

**Neuralgia of the Genitalia and Rectum.**—These varieties are not met with frequently. The former is sometimes a symptom of stone, prostatic disease, or stricture, and in women ovarian and uterine neuralgias are generally hysteric manifestations. Coccydynia, unless of traumatic origin, is almost solely found in women. The pain in the region of the coccyx is excruciating at times, and may even call for operation.

**Visceral Neuralgia.**—As implied by the name, these forms are neuralgias resident in the various viscera. They most frequently attack the stomach or bowel, and are recognized as colic. Other viscera may also be involved (liver, kidney).

**Treatment of Neuralgia.**—The first requisite in the treatment of neuralgia is to ascertain whether it is due to local or general causes. That of the former class may be caused by a cicatrix, neuroma, aneurysm, neoplasm, or by caries or traumatism; and the treatment must necessarily be directed toward the removal of the cause when possible. When the fault is a general one, the neuralgia may occur either as the immediate result of the systemic disease or remotely, as the result of the altered blood-state (anemia). This is particularly well illustrated

by an attack of malaria, in which it is palpable that success can only be obtained by attention to the underlying cause. It is sometimes necessary to use an analgesic, of which morphin is certainly the best. Its therapeutic value is most decided when the drug is given hypodermically, and if injected directly over the track of the painful nerve (*e. g.* supraorbital branch of the fifth), it not only affords immediate relief, but also obviates recurrences of the painful paroxysms in many instances. It is, however, scarcely necessary to urge the exercise of caution, for the morphin-habit is readily formed in these cases. The following may also be used; anti-pyrin, phenacetin, codein, veratrum viride, aconite, also counter-irritants and vesicants, including the galvanic current. The general tone of the system must be attended to, bad habits prohibited, the state of the bowels regulated, and the eyes examined and corrected for errors of refraction. Rest is a valuable adjunct to any form of treatment. In neuralgia of the upper extremities and in sciatica I have often obtained good results from putting the limb in splints.

Sciatica is often very intractable. If it fails to yield to the salicylates, counter-irritation, leeches, and the rest-treatment of Weir Mitchell may be used, or nerve-stretching, either by flexing the thigh upon the abdomen or, as a last resort, by cutting down upon the nerve itself. Other nerves are sometimes subjected to this method of treatment also, but less frequently than the sciatic. Absolute rest in bed, with the limb kept perfectly still by means of sand-bags or a long splint, always gives relief, and in some cases seems to cure. Alternating hot and cold douches also give great relief in some instances. Deep injections of thein, ether, or chloroform are sometimes used, and even distilled water may give relief when injected into the nerve. The use of guaiacol ( $m_j$ - $i_j$ —0.066—0.1332) in association with chloroform ( $m_x$ —0.666) by this method has yielded very encouraging results in my hands.<sup>1</sup>

## DISEASES OF THE CRANIAL NERVES.

### OLFACTORY NERVE.

THE following morbid conditions have been described in connection with the sense of smell:

(a) *Hyperosmia* or *Olfactory Hyperesthesia*.—The sense of smell is abnormally acute, so that objects, and even persons, can be recognized by this means. It occurs in hysteria and insanity.

(b) *Parosmia* (perverted sense of smell) is due to irritation either of the center or of the nerve-trunk. This perversion may occur for one or for many odors, and is often associated with an obtunding of the normal sense.

(c) *Subjective sensations* of smell are due to the same causes as the above. An olfactory aura may precede an attack of epilepsy. Olfactory hallucinations occur occasionally in the insane.

(d) *Anosmia* or *olfactory anesthesia* (loss of the sense of smell) may be caused by—(1) injury to the peripheral filament by local disease of

<sup>1</sup> "The External and Internal Use of Guaiacol," *Therapeutic Gazette*, Mar. 15, 1895.



the nasal mucous membrane. (2) Injury to the nerve-trunk or bulb, bone-disease, and meningitis. Anosmia may occur during locomotor ataxia. Pungent and powerful odors have been said to cause loss of the sense of smell, due to excessive stimulation. There may be a congenital absence of the olfactory nerves. (3) Centric lesions, as tumors in the anterior part of the temporo-sphenoid lobe. Hughlings Jackson has reported cases of unilateral anosmia associated with aphasia, believed to be due to simultaneous involvement of the outer limb of the olfactory nerve as it passes the island of Reil to reach the center and Broca's region. Opposite unilateral anosmia has been described, due to a lesion in the posterior part of the internal capsule.

In testing the sense of smell it is advisable to use aromatic oils, as they only stimulate the olfactory nerve, while ammonia and such strong substances also stimulate the fifth nerve. It is obviously necessary to make a rhinoscopic examination.

**Treatment** is generally unsatisfactory, though the cause must be removed when possible. When the disturbance is due to some general condition, as hysteria, it may of course be disregarded, as it will improve with the disease.

#### DISEASES OF THE RETINA, OPTIC NERVE AND TRACT.

Since the intra-ocular changes are an index of what is going on in the system in certain blood- and kidney-diseases, syphilis, and brain-troubles, and, indeed, as they sometimes foreshadow coming events, thereby proving a most valuable aid in diagnosis, the following brief description of the lesions, as seen with the ophthalmoscope, is given:

**The Retina.**—Hemorrhage into the retina may be venous or arterial, single or multiple, monocular or binocular. It may be part of a general vascular change; occasionally it occurs during parturition, but more often at the menopause; it may be an indication of renal trouble or of some primary or symptomatic anemia, as in leukocythemia, pernicious anemia, or malaria. Hemorrhage is prone to occur also in depraved nutritional conditions, in purpura, and in scurvy.

More or less complete loss of vision develops in these cases, either suddenly or gradually. If the hemorrhage is superficial, the eye-ground is red and swollen; if deeper, the blood escapes between the fibers of the retina, spreads them out, and assumes a flame-shaped appearance. Mr. Hutchinson thought this was characteristic of gout, but it is now known to be absent in many undeniably gouty cases, and present in others in which no suspicion of gout exists. When multiple hemorrhages occur the irritation consequent thereon causes a turbidity of the retina between the blood-spots ("retinitis apoplectica").

**Retinitis.**—Three forms of this condition are commonly described—the (1) albuminuric, (2) syphilitic, and (3) pigmentary, though Brudenell Carter regards the last named as the only true retinitis, and believes that if the other conditions are inflammatory, they are due to irritation induced by the presence of adventitious deposits.

(1) *Albuminuric retinitis* is probably not a distinct affection, but part of a general fibro-vascular change associated with nephritis. The failure of vision may precede the advent of albuminuria, but more often

the two conditions are coincident. It occurs in chronic nephritis, especially in the interstitial variety.

The retinal changes, according to Gowers, are either *hemorrhagic* or *degenerative*. In the former the arterial blood occupying the interstices between the fibers assumes a striated or feathery aspect, while in the degenerative form white patches of fatty degeneration or deposits of cholesterin are dotted over the fundus; they may also be grouped about the macula lutea, or around the disk. Occasionally the latter appears swollen, owing to the effusion of serum into the fiber-layer.

(2) *Syphilitic retinitis* generally occurs in the later stages of acquired syphilis, and particularly in neglected cases. Failure of vision directs attention to the eye-ground, which is found to have either scattered or uniformly distributed whitish or slightly opalescent filmy patches upon it. The vitreous may be turbid also. Retinitis is far less common than choroiditis or chorio-retinitis.

(3) *Pigmentary retinitis*, as stated above, is believed by Carter to be a true inflammation, attacking the retinal elements themselves and not the fibrous layer. It is essentially a chronic process, usually attacking young adults, and, as a rule, more than one member of a family. It may also occur in inherited syphilis and in low grades of vitality. The affected parts receive a deposit of pigment which specially follows the course of the main arteries. At the same time a circumferential annulus of pigment forms. This gradually encroaches more and more upon the disk, until finally atrophy ensues.

Among retinal affections occur also—

(a) **Toxic Amblyopia**.—This is due, as a rule, to tobacco or alcohol, and more rarely to certain drugs or lead-poisoning. Failure of vision is gradual and progressive, though it rarely reaches absolute blindness. The center of the field is chiefly affected, and a central scotoma for red and green exists; this is said to be caused by a chronic neuritis beginning in the fibers that are distributed to the macula lutea.

(b) **Hemeralopia**, or *day-blindness*, may either be functional or a symptom of some retinal affection—*e. g.* hyperesthesia or albinism. Objects can either not be seen at all or only indistinctly during the day or in a strong artificial light; but at night vision is excellent.

(c) **Nyctalopia**, or *night-blindness*, may either exist without apparent cause or it may be symptomatic. It occurs rarely during the course of wasting diseases, as in scorbutus, albuminuria, and anemia. Far more commonly it exists in connection with syphilitic retinitis or choroiditis, separation of the retina, or some congenital defect, and is always present in retinitis pigmentosa. In this condition vision may be normal during the day or in a strong artificial light, but after nightfall or in a darkened room objects can be seen only with difficulty or not at all.

Hemeralopia and nyctalopia are often used in an entirely opposite sense from that employed here, but the definitions given are etymologically correct, and have been adopted in the nomenclature of the Royal College of Physicians of England.

**Optic Nerve**.—Three distinct pathologic conditions of the nerve exist—*viz.* (1) Perineuritis, (2) Choked disk, and (3) Neuritis. They may merge into one another, and after lasting some time may lead to (4) Atrophy and complete blindness.

(1) *Perineuritis* is met with in meningitis, and was suggested by Bouchut as a valuable diagnostic sign in obscure cases. It is also found in certain cases of insolation, and may occur in any fever in which impairment of vision is a sequel. The sheath of the nerve is supplied by the blood-vessels of the pia, while the nerve itself derives its blood-supply from the anterior cerebral artery; therefore in perineuritis, in the early stages at least, the disk remains normal, but is surrounded by a zone of congestion and inflammation. If the action has been severe or prolonged, either the direct pressure or that due to the contraction of the inflammatory material causes partial atrophy of the nerve and consequent interference with the vision.

(2) *Choked disk* is almost always bilateral, and occurs in cases of intracranial granulomata or tumors, also in Bright's disease and syphilis. The disk is at first merely congested; soon, however, both the arterial and venous circulations are interfered with, and especially the return venous flow; then exudation of serum takes place. Sometimes secondary inflammatory changes follow. In the early stages vision is not impaired, but as the exudative elements contract, the interference with the circulation becomes more exaggerated, and in time atrophy of the disk supervenes. Should the process be arrested, the retinal dropsy subsides, and it will then be seen that the vessels are thickened and tortuous, and stand out in relief near the margins of the disk. White patches of atrophy may be scattered over the fundus. When the inflammation and a dropsical effusion into the disk exist simultaneously, it is difficult or impossible to differentiate the condition from primary or descending optic neuritis.

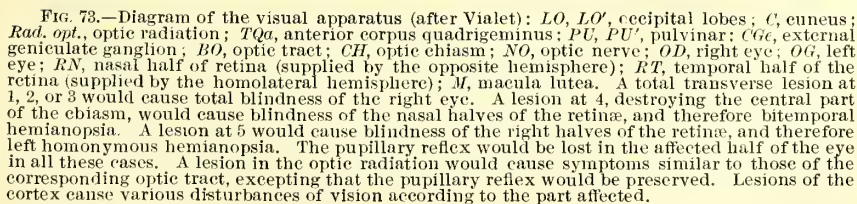
(3) *Neuritis*.—The optic nerve derives its blood-supply from the anterior cerebral artery. Evidently, then, in cerebral hyperemia (arterial) from any cause we have an increased injection of the disk, but no venous engorgement; hence there is no dropsy and no tortuosity of the veins. Inflammation may begin in the disk or descend from above, giving rise to plastic deposits on the retina. Sight is early interfered with, owing to involvement of the conducting fibers, which atrophy in time unless the condition ceases. Then the disk appears white, and the vessels show upon it as thin filaments. This condition is met with in syphilis, Bright's disease, intracranial tumors, and rarely in anemia and lead-poisoning; it may be an advanced stage of perineuritis or choked disk.

(4) *Optic Atrophy*.—This may occur as an hereditary affection known as *Leber's disease*, which chiefly attacks young males, or it may occur during the course of locomotor ataxia, certain toxemias, and diabetes. It may also be brought about either by conditions that produce brain-disease or as a result of the cerebral disease itself.

In any case there is alteration of the field of vision, color-perception is abnormal, and there is more or less dimness of sight. In the hereditary form the disk is less white than in the other, and the vessels are almost normal in appearance (Fig. 73).

**The Optic Tract.**—The lesions of the optic tract are important rather on account of their situation than their nature. They may exist without corresponding changes in the retina, although when they have lasted for a long time there is usually some consecutive atrophy resulting from a descending degeneration of the optic nerves. Lesions of the chiasm





usually affect the decussating fibers, causing blindness of the nasal halves of the retina, and, in consequence, temporal hemianopsia. This condition occurs in basal tumors especially of the hypophysis, and has therefore been observed in acromegaly, in tuberculous basal meningitis, and in hydrocephalus. Lesions of either optic tract, if complete, causes homonymous bilateral hemianopsia; if incomplete, there is irregular disturbance of the visual field, sometimes bilateral, sometimes unilateral. It may be involved in hemorrhage, tumors, softening or basilar meningitis; ordinarily other structures are also involved, giving rise to symptoms of focal disease. Lesions anterior to the anterior corpora quadrigemina usually cause more or less destruction of some of the other cranial nerves, with the production of ocular palsies, or disturbances of the other special senses, or anesthesiæ or neuralgias of the face. A very valuable sign, that, however, cannot always be elicited, is the failure of the pupil to contract when light is thrown upon the blind half of the retina. This is explained by supposing that the pupillary reflex center is situated in the anterior corpus quadrigeminus. If the lesions affect the optic thalamus or the internal capsule, hemiplegia and hemianesthesia are also often present or may form the most important symptoms. Lesions posterior to the anterior corpora quadrigemina produce hemianopsia without disturbance of the pupillary reflex. These lesions are divided into two groups, the cortical and the subcortical and they may be of two varieties, either irritative or paralytic. The irritative lesions give rise to hallucinations of sight, which may vary from the scotomata of migraine to most complex visions. Paralytic lesions ordinarily lead to hemianopsia. Occasionally curious symptoms are produced, the visual field being sometimes irregular, whilst at others only certain elements of sight are affected, cases having been reported in which the hemianopsia only involved the recognition of colors, not of form. In all these cases the pupillary reflexes are not affected. Bilateral lesions do not always lead to total blindness: sometimes the macula lutea escapes and the patient is able to see only by direct fixation. Occasionally a single lesion will produce total blindness in one eye, but this is rare, and no satisfactory explanation has been found to account for it. Cortical lesions are those involving the occipital lobe. The center of visual perception appears to be in the cuneus and calcarine fissure; if this is destroyed, blindness occurs. The center for the recognition of the object seen is apparently upon the convex surface of the occipital lobe, probably in the second and third convolutions, but it may extend also into the temporal lobe. When this is destroyed the patient can see objects, but fails to recognize them; this is called *mind-blindness*; if total, this is the result of a bilateral lesion. Hemianopsia is very frequently merely a temporary symptom, and as such it may occur in uremia, apoplexy, migraine, and certain intoxications, especially that of lead. It may also occur in brain-tumor, and disappear if the pressure is relieved, as by trephining. It is a permanent symptom only when the visual tract has been involved by some destructive lesion. If the patient is perfectly conscious and intelligent, it is not difficult to recognize it; nevertheless, its presence can often be detected in young children and in those who are only partially conscious or unable to speak. This can be accomplished by taking a bright object, placing it behind the head, and then bringing it

forward slowly, first on one side and then on the other. It will then be noted that the patient perceives it on the hemianopsic side only when it has been brought to the middle line, whilst when moved on the other side the eyes will turn toward it when it is still a considerable distance from this point. Another method is to bring a blunt object (a wisp of cotton) very nearly in contact with the cornea, first on the one and then on the other side of the median line. The palpebral reflex will occur upon the normal side whilst the object is still some distance away; on the blind side only when it has come in contact with the conjunctiva (see Fig. 73).

#### DISEASES OF THE MOTOR NERVES OF THE EYEBALL (THIRD, FOURTH, AND SIXTH).

The extrinsic ocular muscles are supplied by these three nerves, while the intrinsic are supplied by the third and the sympathetic.

**I. The motor oculi, or third nerve,** is purely motor, and supplies all the muscles of the eye except the superior oblique and external rectus, and controls in part also the ciliary muscle and the sphincter of the iris. Its apparent origin is from the inner side of the crus cerebri just anterior to the pons. It can be traced through the crus, however, to its deep origin in a nucleus beneath the corpora quadrigemina, situated in the floor of the aqueduct of Sylvius. Above the crus it pierces the dura, passes between the two clinoid processes of the sphenoid bone, along the outer wall of the cavernous sinus, where it receives some filaments from the cavernous plexus of the sympathetic; it then divides into two branches that enter the orbit through the sphenoid fissure. The superior and smaller division supplies the superior rectus and levator palpebræ superioris, while the inferior and larger branch subdivides into three portions, one going to the internal rectus, another to the inferior rectus, and the third to the inferior oblique.

Lesions of the third nerve result in (1) spasm or (2) paralysis.

**Spasm** rarely if ever occurs in all the muscles simultaneously. Any muscle may be affected, but the internal rectus and levator palpebræ are specially liable. The condition is met with in meningitis, hypermetropia, and hysteria; also in nystagmus, in which the spasm is clonic and bilateral; it also occurs in albinism, occasionally in coal-miners, or it may be congenital.

When the levator palpebræ is affected (*lagophthalmus*) inability to close the eye results. Stimulation of the center or nerve may cause contraction of the pupil (*myosis*), as occurs in locomotor ataxia. The same result is brought about by paralysis of the sympathetic.

**Paralysis.**—Usually the nerve is involved as it passes through the dura or at the orbital foramen by some inflammatory process, rheumatic or syphilitic, or it may be the result of meningitis. Pressure due to a gumma or other tumor or to an aneurysm, and sometimes traumatism will bring about the same result. Paralysis may also be due to a neuritis the result of diphtheria or some other infectious disease, toxemia, or locomotor ataxia. More rarely the nucleus is damaged by hemorrhage or inflammation. In such cases, however, owing to their intimate relationship, the nuclei of the other eye-muscles will usually be involved,



giving rise to general ophthalmoplegia. When the intra-ocular muscles alone are affected it signifies a central lesion.

Relapsing and recurring palsy are two peculiar varieties. The former occurs chiefly in syphilitic subjects. One nerve becomes affected and partially recovers; the other one then becomes paralyzed, and partially recovers, relapses, and so on. The internal muscles may be involved.

Recurring or periodic palsy, the *migraine ophthalmique* of Charcot, is a rare form. It occurs in both sexes, but women are especially susceptible. It may begin in infancy and recur at intervals for years, the attacks being periodic, lasting a few days to six or eight weeks, and ending in complete recovery. They may be precipitated by some emotional disturbance, by menstruation, or by exhaustion. Their exact nature is not understood, but they resemble migraine in that there is severe headache or pain, usually over one eye, and in their association with vomiting.

Generally paralysis of the extra-ocular muscles is partial, and the symptoms will vary according to the muscles affected. When they are all involved there are ptosis, divergent strabismus, diplopia, and contracted pupil, with loss of the light-reflex and accommodation.

*Intra-ocular Paralysis.*—(a) *Cycloplegia*, or ciliary muscle-paralysis, gives rise to a loss of the power of accommodation, so that “far-sight” is good, while “near-sight” is blurred and indistinct. This can be corrected by a convex glass. Bilateral cycloplegia is usually due to a nuclear lesion. It occurs sometimes in diphtheria and in tabes dorsalis.

(b) *Iridoplegia*.—The pupil may be dilated (*mydriasis*) from palsy of the sphincter or spasm of the dilator, or it may be contracted (*myosis*) from the antithesis of the above.

The iris has three actions—two reflex and one associated: First, a reflex contraction of the sphincter on exposure of the eye to light; second, a reflex dilatation of the radiating fibers on stimulation of some cutaneous nerve; and, third, a contraction on accommodation, usually, but not necessarily, associated with convergence (Gowers).

First, light-reflex iridoplegia. The iris reflex is lost in locomotor ataxia, and may be also in syphilis. Accommodation and convergence are, however, usually preserved (*Argyll-Robertson pupil*). When these also are lost the condition occurs to which Jonathan Hutchinson gave the name *ophthalmoplegia interna*.

In testing this reflex care must be taken to avoid the contraction of accommodation. The patient should look at a remote part of the room; then a light is brought suddenly in front of, and three or four feet distant from, the eye. One eye should be examined at a time, the other being covered, but not closed.

Gowers has reported unilateral reflex iridoplegia occurring in tabes. It is extremely rare, however. The reflex path is as follows: the retina, optic nerve, chiasm, both optic tracts, corpora quadrigemina, third-nerve nucleus, third-nerve trunk, ciliary ganglion, and the ciliary nerves.

Second, skin-reflex iridoplegia. Normally, painful stimulation of the skin of the neck causes reflex dilatation of the pupil, the afferent impulse being carried along the sympathetic. In locomotor ataxia

myosis often exists. In such cases Erb showed that the skin-reflex was lost (*spinal myosis*).

Third, accommodation iridoplegia, in which the power of accommodation is lost. The pupil does not become smaller when looking at near objects. Westphal and Piltz have recently discovered independently, that in certain pathological conditions the pupil contracts strongly upon closure, or attempted closure against resistance, of the eyelids. This reflex occurs most constantly in general paresis. Its exact significance is not known.

II. **The fourth nerve, or patheticus**, the smallest cranial nerve, supplies the superior oblique muscle. Its superficial origin is to the outer side of the crus cerebri, just in front of the pons. The fibers can be traced backward to the valve of Vieussens, in the substance of which it decussates with its fellow. Its deep origin is in a nucleus in the floor of the aqueduct of Sylvius, immediately behind and in close connection with the third-nerve nucleus. After piercing the dura mater the nerve runs along the outer wall of the cavernous sinus and enters the orbit through the sphenoid fissure. Since the superior oblique muscle directs the eyeball downward and rotates it, paralysis causes defective downward and inward movements, and consequent diplopia with inclination of the head forward and to the sound side. When occurring alone it is probably due to a nuclear lesion.

III. **The sixth nerve, or abducens**, has its deep origin in the floor of the fourth ventricle in close proximity to the seventh-nerve nucleus. Its superficial origin is from the lower part of the pons, in the groove between it and the medulla. Emerging, it pierces the dura, runs in the cavernous sinus, and enters the orbit through the sphenoid fissure to supply the external rectus. Owing to its long course, this nerve is specially liable to injury, usually from pressure due to tumors or from syphilitic or other forms of meningitis. Paralysis of the muscle causes convergent strabismus, owing to an inability to rotate the eye outward and consequent diplopia. In nuclear lesions the external rectus of the same side and the internal rectus of the opposite side are paralyzed, conjugate deviation resulting, the eyes being directed away from the side of the lesion. This is due to the fact that the sixth nerve gives off a twig that runs to that region of the opposite third-nerve nucleus governing the internal rectus.

This muscle is not wholly controlled by the sixth nerve, however, for in nuclear lesions of the latter no degenerated fibers are found in the third nerve; and, further, when the eye with the paralyzed external rectus is covered the opposite internal rectus will act, though less readily than normally.

### **General Symptomatology of Paralysis of the Eye-muscles.**

—Loss of power in the ocular muscles is indicated by five kinds of symptoms (Gowers): (1) *Limitation of Movement*.—The amount of limitation in the movement of the eyeball is in direct ratio to the degree of palsy. In complete palsy the globe is ultimately fixed, owing to contraction of the unopposed muscle. In partial paralysis, as the limit of movement is approached the motion is often jerky (*paralytic nystagmus*).

(2) *Strabismus*.—Owing to defective movement the axes of the eyes

do not correspond. "The deviation of the axis of the paralyzed eye from parallelism with that of the sound eye is termed the primary deviation."

(3) *Secondary Deviation*.—"If the sound eye is prevented from seeing the object, and the patient looks at this (is made to 'fix' it) only with the affected eye, the sound eye is moved still farther in that direction, and hence the deviation of the visual axes is increased. This is called the 'secondary deviation,' and depends on the fact that two muscles normally acting in unison are equally stimulated (innervated) for any given movement. When one is weak, the amount of nerve-force employed to move the sound eye acts equally on the impaired eye, and hence the over-action. In paralytic strabismus fixation with the sound eye shows the primary deviation, while fixation with the affected eye reveals secondary deviation. In ordinary strabismus due to spasm this does not hold good; it matters not which eye is used, deviation remains the same."

(4) *Erroneous Projection*.—"We judge of our relation to surrounding objects by the position of the eyeball as indicated to us by the degree of stimulation necessarily brought to bear on the ocular muscles. When one of these muscles is weak, the additional stimulation (innervation) necessary to move it in fixing an object impresses us with the idea that it is really farther away than is actually the case, and in attempting to touch it the finger goes beyond. This erroneous projection, or interference of visual sense-impressions, causes a disturbance of equilibrium and gives rise to vertigo, which has been named "ocular vertigo."

(5) *Double Vision*.—"This is not due alone to a difference in the axis of vision, causing images on non-corresponding portions of the retina, but also to the erroneous projection. "If the patient looks with both eyes, the field of the unaffected eye, being normally projected, does not correspond with the field of the affected eye; the images formed in the two eyes are mentally referred to different positions; objects are seen double" (Gowers). The "true image" is that one formed in the sound eye, while the retina of the affected eye receives the "false image."

*Homonymous* or *simple diplopia* is that in which the false image appears on the "same side of the other as the eye by which it is seen." This is due to paralysis of an abductor muscle—*convergent strabismus*. *Crossed diplopia* occurs in divergent strabismus, the result of paralysis of an adductor. The false image appears to be on the other side of the real object—*i. e.* toward the sound eye.

Gowers' mnemonic is, "When the visual lines (prolonged ocular axes) cross, the diplopia is not crossed."

**Ophthalmoplegia**, a paralytic condition of the eye-muscles, may be partial or complete. Either the internal or the external muscles may be involved, constituting *ophthalmoplegia interna* or *externa*, and, when both are affected, total ophthalmoplegia. The lesions may be nuclear or peripheral. Pressure due to neoplasms, gummata, aneurysms, or basilar meningitis may produce it, or it may follow diphtheria. It also occurs in general paralysis, progressive muscular atrophy, and locomotor ataxia. It may be (a) of sudden onset, due to some vascular disturbance; (b) acute—the *polio-encephalitis superior* of Wernicke—develop-



ing in a few days or weeks; or (*c*) chronic. In the latter case symptoms of bulbar palsy are apt to be present also. Von Graefe has described a form of bulbar palsy limited to the ocular nuclei under the name of *progressive ophthalmoplegia*.

The **symptoms** vary necessarily according to the muscles involved.

The **treatment** consists in the removal of the cause when possible. In inflammatory cases counter-irritation is employed by blisters placed on the temples, behind the ears, or at the occiput, or by leeches. Internally, the salicylates, mercury, iodids, and general tonics are useful. Rarely a case will recover spontaneously. Electricity is probably of little value. The diplopia, unless it can be obviated by a suitable lens, should be met by means of an opaque glass.

#### DISEASES OF THE FIFTH NERVE.

The trigeminus nerve has an extensive origin from the floor of the fourth ventricle. It supplies with sensation the whole region innervated by all the other cranial nerves except the first and second. It resembles a spinal nerve in that it has two roots, a motor and sensory, and on the latter a ganglion (*Gasserian*). From the latter arise three sensory branches—viz. the ophthalmic, superior maxillary, and inferior maxillary. A motor root joins the last named, the largest branch of the fifth nerve.

Morbid conditions of the fifth nerve cause sensory, motor, or gustatory symptoms. The lesion may be—(1) Pontine hemorrhage, softening, sclerosis, or tumor. (2) Disease or injury at the base of the brain—*e. g.* meningitis, gumma or other tumor, caries of bone. (3) Disease or injury of the branches, as neuritis, pressure due to aneurysm of the internal carotid or to a tumor in the pituitary or sphenomaxillary region, orbital cellulitis, and punctured wounds of the mouth and nose. (4) Rarely fracture of the skull.

**Symptoms.—Sensory Portion.**—In the irritative stage the chief feature is pain; this may be shooting, boring, or burning in character. Tenderness along the course of the nerve and hyperesthesia may also exist. Later anesthesia develops in the mucous membrane of the nose, mouth, lips, tongue, and, in some cases, of the hard and soft palate also. Muscular movements are slower than normally, due to sensory interference.

The secretions are often increased, though at first they are lessened; hence the anosmia, due to dryness of the nasal mucosa. Loss of sense of taste may also occur. Other trophic changes are—inflammation and ulceration of the gums, looseness of the teeth, and inflammation of the eye. Corneal opacities, ulceration, sometimes perforation, and finally complete destruction of the eye—neuro-paralytic ophthalmia—are noted. This is especially apt to occur when the Gasserian ganglion is involved. Painful and intractable herpes may develop. Hemifacial atrophy may result from disease of the fifth nerve (Mendel).

**Motor Portion.—Spasm or Paralysis.**—Partial or complete inhibition of the movement of the muscles in the region supplied—*i. e.* those of the jaw, the masseter, temporal, pterygoid, mylo-hyoid, and the posterior belly of the digastric. The degree of palsy can be ascertained by

placing a finger on each masseter or temporal muscle while the patient alternately opens and forcibly closes the mouth. In external pterygoid paralysis movement toward the sound side is impossible, and on depression of the lower jaw it deviates toward the affected side. Ultimately wasting of the muscles, with deformity, takes place.

The spasm (the so-called "masticatory spasm" of Romberg) may be tonic or clonic. In tonic spasm—trismus or lockjaw—the jaw is firmly set and the muscles are hard, rigid, and sometimes painful. This occurs in tetanus, in certain cases of tetany and hysteria, in caries of the teeth, occasionally after exposure, and in irritative centric or peripheral lesions. Clonic spasm is more or less continuous or intermittent. The former consists of short, quick, vertical or rarely lateral movements (*e. g.* chattering of the teeth), usually associated with some other condition, as paralysis agitans, general convulsions, and the like, or it may exist alone, especially in women late in life. The intermittent form is rare and occasionally occurs in chorea. Contractions are single, forcible, and are separated by some little time. The tongue and cheeks may be bitten in the attack.

**Gustatory Portion.**—*Symptoms* referable to this portion are not always present in disease of the fifth nerve. There may be a loss of taste without sensory disturbance, or *vice versâ*, or both may exist contemporaneously. Lesions of the nerve-root or middle-ear disease may cause it, but pontine lesions, as a rule, do not. A perverted sense of taste—*parageusia*—may be present in hysteria and insanity. Increased sensitiveness—*hypergeusia*—and subjective sensations of taste may result from irritative lesions, and the latter may precede an attack of epilepsy (as an aura).

The **diagnosis** is not difficult as a rule. Anesthesia in the area supplied by the nerve, with loss of taste, is fairly conclusive. Spasm may be simulated in cases of rheumatism or rheumatoid arthritis involving the temporo-maxillary articulation.

**Treatment.**—The underlying cause should be attacked when possible, and mercury, the iodids, and the salicylates should be administered in specific cases and in those due to exposure. Analgesics, and even opiates, may be necessary. Sometimes vigorous counter-irritation is of value. Attention must be paid to the condition of the general system. The battery may be tried, preferably with the faradic current, or by means of electricity short and extremely rapid blows may be made over the nerve.

#### DISEASES OF THE SEVENTH OR FACIAL NERVE.

The nucleus of this nerve in the floor of the fourth ventricle is in relation with those of the sixth, eighth, and twelfth nerves. Like the spinal nerves, it has an upper and lower neuron or motor segment, the former extending from the cortical center in the lower Rolandic region to the nucleus, while the latter runs from the nucleus to the periphery. Lesions may involve any part of the tract, producing either spasm or paralysis.

**Spasm.**—This may be idiopathic or organic, and either general or

partial, affecting only the orbicularis palpebrarum (*blepharospasm*). It is sometimes called *tic convulsif* or mimic spasm.

**Pathology.**—The commonest cause appears to be some psychic disturbance. Next in frequency are peripheral irritations, and particularly those that involve the trigeminus, as carious teeth, conjunctivitis, or some nasal irritation. Less frequently irritation in some other part of the body, as intestinal parasites or uterine disease, may be the exciting cause. Finally, there may be lesions in any part of the motor tract supplying the face, either in the cortex (meningeal tumor, exostoses, or focal softening), in the facial nucleus in the medulla, or along the course of the facial nerve (aneurysm or atheroma of the vertebral artery). Morbid changes in the nerve itself or in the muscles have not been observed.

**Etiology.**—As in other functional diseases, neuropathic heredity plays an important part. Sometimes, indeed, tic convulsif has existed in a parent or has occurred in several children of the same family. Emotional disturbances have frequently been the exciting cause; besides these, there are the various reflex irritations. The organic causes are irritative lesions, situated in some portion of the facial motor tract. According to Gowers, the functional form occurs only in adult life. It occasionally follows facial palsy.

The **symptoms** of the disease include, first, the *spasm*: this is usually a sudden clonic convulsion of the muscles of one side of the face, with closure of the eyelids and retraction of the angle of the mouth. Rarely there are associated movements of the palate and eyeballs. The spasms may be single or they may occur in groups frequently repeated, or recur constantly at more or less irregular intervals. Less frequently the contraction may be tonic in character, lasting several seconds or even minutes. These forms are frequently associated with clonic spasms. Ordinarily the spasm is painless, though there are certain sensitive points, as where the branches of the trigeminus issue from the skull, and particularly over the supraorbital foramen. Sometimes there is also *tinnitus aurium*. *Taste* and the muscles of the palate are rarely affected. The immediate exciting cause of an attack may be fatigue or excitement, or it may occur as an associated movement, as in a case that I observed, in which spasm always accompanied the beginning of speaking.

The **diagnosis** is usually easy. It may be occasionally confounded with *chorea*, especially when the latter is chiefly localized in the face, or with *athetosis* due to infantile brain-lesions. Recognition of the cause is often very difficult, and a careful examination of the whole body should be made for any possible source of irritation.

The **prognosis** is extremely unfavorable for cure, since only in cases of recent occurrence, and with a distinct source of peripheral irritation, is permanent recovery likely. Ordinarily, the disease, even if commencing in a mild form, gradually progresses to the most severe type, the intervals between the attacks become shorter, and the attacks themselves more severe.

The **treatment** consists in the removal of any source of irritation and the application of electricity, particularly the mild galvanic currents, with the anode over the sensitive points. Operative interference, as stretching the facial nerve (which rarely produces any result unless paralysis ensues) or cutting the tendons of the facial muscles, may be tried;



and finally, use of antispasmodics, as conium, gelsemium, morphin, and the bromids, may give temporary, but rarely permanent, relief.

**Paralysis (Bell's Palsy).**—Depending on the seat of the lesion, we have—(a) supra-nuclear, (b) nuclear, and (c) infra-nuclear palsy. The following table presents the general differences between upper and lower neuron palsy :

SUPRA-NUCLEAR PARALYSIS.	NUCLEAR AND INFRA-NUCLEAR PARALYSIS.
The upper part of the face is not affected, the muscles of the angle of the mouth being chiefly concerned.	All parts of the face involved, including the orbicularis and frontalis.
Voluntary movements are more impaired than the emotional.	Voluntary and emotional movements equally affected.
All reflex movements are normal.	All reflex movements are lost.
Electric reaction is normal, or only slightly impaired to both galvanic and faradic currents.	Reactions of degeneration are present.
There is no wasting.	Wasting is present.

(a) *Supra-nuclear paralysis* is generally associated with hemiplegia, the palsy of face and limbs being on the same side—*i. e.* opposite the lesion, which may consist of a hemorrhage, tumor, abscess, softening, or which may be the result of injury, and may be situated in the cortex, corona radiata, or the internal capsule. When the cortical face-center is alone involved, the limbs escape (*monoplegia facialis*). This form is rare.

(b) *Nuclear paralysis* is due to hemorrhage, tumor, or softening at the site of the nucleus in the medulla. It may also result from an attack of diphtheria, and very rarely occurs in cases of antero-poliomyelitis. As already noted, the symptoms are similar to those of infra-nuclear paralysis.

(c) *Infra-nuclear paralysis* is caused by pressure on the nerve at the base of the brain by tumors, meningitis, aneurysm, or hemorrhage. In the Fallopian canal the nerve may be damaged by bone-disease or some form of otitis. This is the seat, too, of the so-called “rheumatic neuritis,” the result of exposure.

Fracture of the base of the skull or injury to the nerve as it emerges from the stylo-mastoid foramen may result in facial palsy. *Diplegia facialis* is rare, but may be caused by a single lesion in the pons, where the facial paths cross, or by two lesions, one on either side. The causes enumerated above, when bilateral, beget double facial paralysis.

Lesions in the lower part of the pons may result in crossed hemiplegia, the fibers being involved in their course between the nucleus and the point of emergence of the nerve, the side of the pons. The face will be paralyzed on the same side as the lesion, since this latter is below the decussation of the facial tracts, and involves the outgoing nerve, together with opposite hemiplegia. In alternate or crossed hemiplegia the facial palsy is of the infra-nuclear type, while in ordinary hemiplegia the supra-nuclear type is met with. Certain symptoms of nerve-irritation may precede the actual palsy or may be concomitant, such as slight pain and tenderness, some swelling in front of the ear, muscular twitching, and occasionally vertigo.

*Symptoms.*—The affected side is immobile and expressionless, and the

normal lines are diminished or abolished. This is seen most markedly in those above middle life. The eye cannot be closed, owing to drooping of the lower lid, and, as the tears are not directed into their proper channel, the eye waters. Voluntary and emotional movements are lost. Whistling and smoking are performed with difficulty, if at all, and food collects between the teeth and cheek of the affected side, owing to paralysis of the buccinator; in drinking the patient inclines the head to the sound side to prevent escape of the liquid from the corner of the mouth. The dilator naris is paralyzed; hence sniffing is interfered with, and the sense of smell is lowered on that side.

When the tongue is protruded it seems to be drawn toward the palsied side. This is not the case, however, the effect being due to contraction of the unopposed muscles on the sound side. All reflex movements are lost. The palate is not affected, and sensation is not impaired. When the nerve is involved between the *intumescencia gangliiformis* and the origin of the chorda tympani, taste is lost in the anterior part of the tongue. When other parts of the nerve are diseased, taste is not interfered with, unless an ascending—or, more rarely, a descending—neuritis develops. Hearing may be increased, owing to paralysis of the stapedius, with consequent unopposed action of the tensor tympani. In ear-disease and in disease of the base of the brain, involving both facial and auditory nerves, hearing is lessened. Some degree of wasting takes place in the affected muscles, and both quantitative and qualitative electric changes quickly follow the palsy.

The duration of an attack varies from a few days to several months or a year, and in rare cases it is permanent. The onset is usually prompt, and the acme of the attack may be reached in from a few hours to a couple of days.

*Diagnosis.*—From the table previously given it will be easy to differentiate supra-nuclear from infra-nuclear palsy. In cases of long standing, when contractures have taken place, owing to the furrows thus produced the affected side may be taken for the sound side, but on getting the patient to whistle the true state of affairs will manifest itself.

*Treatment.*—Search for the cause. If ear-disease is present, make provision for free drainage; if syphilis, give iodid of potash, mercury, or both. In cases due to cold, the so-called rheumatic palsies, counter-irritation is especially called for, and cantharidal collodion, fly-blisters, or the actual cautery behind the ear or over the occiput are very useful. The bowels should be freely opened, and diaphoretics or hot baths and alkaline diuretics administered; in the inflammatory stage small doses of mercury are of value, and later mercuric iodid or general tonics. Galvanism may be employed to stimulate the nerves and to help in maintaining the tone of the muscles. When contractures threaten in late cases the use of electricity should be dispensed with.

#### DISEASES OF THE AUDITORY NERVE.

The eighth nerve has its deep origin in the medulla. The center is connected by fibers with the cerebellum, probably by means of an equilibrium mechanism. The auditory fibers decussate in the region of the nuclei, passing in the posterior extremity of the internal capsule to the

opposite hemisphere. The cortical center is in the middle of the first temporo-sphenoid convolution. Destruction of that of the left side results in *word-deafness*: thus, spoken words may be heard, but are not recognized as such. This is not a common condition. Rarely the auditory tract may be involved between the cortex and the nucleus. The nerve may be implicated at the base of the brain by tumors, aneurysms, hemorrhage, meningitis, and traumatism. Erb has described a primary nerve-degeneration in *tabes dorsalis*. Disease may attack the labyrinth, either primarily or secondarily to ear-disease. Drugs—quinin, apiol, salicylates—may cause deafness similar to the labyrinthine variety. In anemia and in other conditions in which the general health is below par, also in hysteria, hearing may be affected. The lesions give rise either to an increased or diminished sense of hearing:

(a) *Hyperacusis*, in which certain or all sounds are intensified. Paralysis of the stapedius muscle causes low notes to be heard with great intensity. Auditory hyperesthesia may also occur in hysteria or during the course of cerebral or general disease.

(b) *Dysacusis*—difficult hearing—may be due to middle-ear disease, or it may exist as a “nervous deafness,” the result of labyrinthine or nerve-disease. These may be differentiated by means of the tuning-fork. Normally, air-conduction is better than bone-conduction, and if in a deaf person a tuning-fork can be heard vibrating longer when held against the skull-vault or temporal bone than in front of the ear, there is some impairment of conduction in the meatus or middle ear. When the patient is deaf, and yet the normal relation is maintained between air- and bone-conduction, the labyrinth or the nerve is at fault.

(c) *Tinnitus aurium*—irritation of the auditory nerve—a condition in which subjective sounds occur, such as whirring, buzzing, ticking, or ringing in character. In certain subjects they are worse at night than during the day, and at times they are paroxysmal; as a rule, in any case they are intensified when the general system is below par.

Tinnitus may be caused by anemic or depraved nutritional states, intra-cranial aneurysm, pressure on the cervical sympathetic by enlarged glands, tumor, or aneurysm, impacted cerumen, otitis media, labyrinthine disturbance, blows upon the head, excessive auditory stimulation, loud noises, or it may occur during an attack of migraine or as an epileptic aura. In a neurasthenic individual the subjective noise, no matter what the cause, will be accentuated. The more complex and elaborate the sound, the greater the probability of its being of central origin.

**Treatment.**—Careful search must be made for the cause of any of these morbid conditions just described, and when practicable they should be removed. The system should be brought into as good a condition as possible. In hyperesthesia bromids occasionally avail. In dysacusis little can be done when the cause is labyrinthine. The same is true when the nerve or its centers are involved. Counter-irritation and electricity may be tried externally, and iodids internally. These measures should be employed in tinnitus, but with more hope of relief; in addition, sedatives are generally called for, and even morphin may be necessary in paroxysmal attacks.



## MÉNIÈRE'S DISEASE.

**Definition.**—An aural or labyrinthine vertigo—originally described by Ménière in 1861; the cardinal symptoms are vertigo, deafness, noises in the ear, and sometimes vomiting.

**Pathology.**—There may be an inflammation or atrophy of the nerve-endings. There are also changes in the labyrinthine membrane from any cause or from hemorrhage.

**Etiology.**—Ménière's disease is most common after thirty, and is rarely met with before that age. It is twice as common in men as in women. The precise lesion is labyrinthine, and is the result of exposure, gout, syphilis, senile change, congestion, and, more rarely, hemorrhage. Any cerebral disturbance or gastric or other irritation is apt to induce an attack.

**Symptoms.**—Vertigo is present, and varies from an extremely slight transient attack, and one that is entirely subjective, to one of almost explosive violence. The patient may have a sensation of having been struck, and then of falling heavily to the ground. The slight form may be continuous with more or less frequent severe attacks, or a complete intermission of days, weeks, or months may transpire. The attacks may arise without apparent cause, or as a result of a blow or even a sudden movement, and occur during both working and sleeping hours. The giddiness, when severe, causes nausea and vomiting, and, if prolonged, bile is vomited as in ordinary bilious attacks. When the attack is very acute momentary unconsciousness supervenes. Nystagmus and diplopia may occur during an attack. Tinnitus and deafness usually exist together, the former being constant, but of slight degree, and possibly worse during an attack; it may be entirely absent between the attacks. The latter (nervous deafness) is constant and of varying severity in different individuals.

**Diagnosis.**—The occurrence of vertigo and tinnitus in a person with more or less nervous deafness, with or without gastric symptoms, establishes the diagnosis. The tinnitus and the character of the deafness usually suffice to distinguish this from other forms of vertigo. In epilepsy with auditory auræ the period of unconsciousness is generally much longer, and on regaining consciousness the patient is dull and drowsy for some time. It is possible also, as a rule, to elicit a history of convulsions.

**Prognosis.**—In some cases the condition grows progressively worse until deafness supervenes, when it ceases. Often, however, arrest or improvement, or even complete recovery, may be secured. In heart-disease the shock may prove fatal, and in the very acute but, fortunately, rare cases the prognosis is always bad.

**Treatment.**—Counter-irritation over the mastoid process and the internal use of bromids to lessen the morbid sensibility will prove valuable. The emunctories must be gotten in good condition, and any underlying disease, as syphilis or gout, must be treated. Charcot suggested the use of drugs that produce tinnitus—quinin, for instance. The cases were worse at the time, but some of them seemed to improve subsequently. Gowers employs sodium salicylate in 5-grain (0.324) doses, thrice daily, believing that more good arises when such drugs are given

in moderation. Apiol might be tried in this connection. Nitroglycerin and the nitrites are sometimes of value in cases associated with arteriosclerosis.

#### DISEASES OF THE GLOSSO-PHARYNGEAL NERVE.

The ninth cranial nerve has its origin in the posterior part of the floor of the fourth ventricle, in close relation with the pneumogastric nerve. Our knowledge as to its function is not exact, both because it is seldom if ever involved alone, and also, on account of its many connections (with the trigeminus, the facial, the pneumogastric, and the sympathetic nerves), it is difficult to say whether the terminal fibers involved represent the functions of its roots or of one of its connections (Gowers).

Its fibers are distributed to the tonsils, the back of the tongue, the soft palate, the pharynx, the Eustachian tubes, and the tympanic cavity. It supplies both motor and sensory fibers, but not those of taste. This nerve is involved in the nuclear degenerations that are spoken of as bulbar palsies. It may be also affected by meningitis or new growths.

#### DISEASES OF THE PNEUMOGASTRIC NERVE.

As already stated, the origin of the tenth cranial nerve is in intimate relation with that of the ninth. It is also continuous below with that of the eleventh, and all three are associated with the center for the hypoglossal nerve. The nerve proper arises from the side of the medulla, and runs on either side of the neck in the sheath of the carotid artery, lying behind that vessel. It enters the thorax in front of the subclavian artery on the right side, and between the subclavian and the carotid on the left; then it courses beside the esophagus, and is distributed to the pharynx, larynx, lungs, heart, esophagus, and stomach, and sends fibers to the intestines and spleen.

The esophageal fibers are both motor and sensory, gastric fibers being chiefly sensory. The vagus is in part the motor nerve of the intestines. It also contains both accelerator and inhibitory fibers for the respiratory center, is the cardiac inhibitory nerve and a vaso-dilator, and is said to contain trophic fibers for the heart and lungs.

**Etiology.**—The nerve may be involved at its nucleus either by hemorrhage or softening. The nuclei of the ninth, eleventh, and twelfth nerves are simultaneously attacked, either wholly or in part, giving rise to a group of symptoms known as *bulbar palsy*. The tenth nerve at its superficial origin may be compressed by neoplasms, aneurysms, and the products of *meningitis*; in its course down the neck it may suffer pressure, or may either be tied in ligating the carotid artery or cut in the removal of a tumor or enlarged glands. Very rarely it may be injured by incised or punctured wounds, or be the seat of neuritis due to exposure or to some toxemia. The morbid conditions of the pneumogastric are best studied by considering the branches of distribution separately.

(a) **Pharyngeal Branches.**—The muscles and mucous membrane of the pharynx are supplied by branches of the pneumogastric and glosso-pharyngeal nerves, constituting the pharyngeal plexus. The pharynx may

be the seat of spasm or paralysis: this is purely a "functional" condition, and usually occurs in hysteric (*globus hystericus*) or in nervous individuals. One of my own patients (a woman) after some domestic trouble became extremely nervous. She complained of increasing difficulty in swallowing, until finally she could scarcely take liquids, this symptom becoming aggravated when any one was watching her. She was cured by the daily passage of graduated esophageal bougies.

Paralysis of the pharynx causes difficulty in swallowing, so that food remains in the mouth instead of being passed into the esophagus. Particles often enter the larynx and give rise to paroxysms of coughing, and at times cause choking. When the soft palate is also paralyzed, the food is regurgitated into the nose. The lesion is generally nuclear, causing bulbar paralysis. The root of the nerve may be involved as it leaves the side of the medulla by meningitis or by pressure from a neoplasm or an aneurysm. Rarely it may be caused by a toxic neuritis.

(b) **Laryngeal Branches.**—The superior laryngeal nerve furnishes sensory fibers to the mucous membrane of the larynx above the vocal cords, and supplies also the crico-thyroid and epiglottidean muscles. The inferior or recurrent laryngeal nerve, which takes its origin in the superior thoracic region, winds around the arch of the aorta on the left side and around the subclavian artery on the right, reaching the larynx by running up between the trachea and esophagus. It is the sensory nerve of the larynx below the vocal cords, also of the entire trachea, and supplies all the muscles of the larynx except those named above. It has been shown that the motor fibers of the larynx come from the glosso-pharyngeal nucleus, the pneumogastric fibers being sensory.

*Spasm of the larynx* is due to over-action of the glottis-closers (the adductors), though some cases described in this category are probably instances of abductor paralysis. The condition is rather rare in adults, but quite common in children (*laryngismus stridulus*), and particularly in rachitic subjects. An attack may also be induced in those predisposed by any form of nerve-irritation or catarrhal condition of the respiratory tract. It may be part of a general neurosis; it is sometimes seen in *tabes dorsalis* (*laryngeal crisis*); and Liveing reports that he has seen it take the place of an attack of migraine. *Spastic aphoria* consists of a spasm induced whenever an attempt to speak is made. Laryngeal spasms occur most frequently at night. Dyspnea is the most striking symptom, and is so intense in some cases that suffocation seems imminent. The patient may be cyanotic. Soon the retained carbonic acid gas causes relaxation, but, as the cords open slowly, the inspiration is accompanied by a crowing sound, and the expiratory sound is harsher than normal.

*Paralysis of the larynx* may be the result of a nuclear degeneration (glosso-pharyngeal), producing chronic bulbar paralysis, as already mentioned; this form may occur in disseminated sclerosis, *tabes dorsalis*, general paralysis of the insane, and in certain toxemias. The paralysis is generally bilateral; rarely it is unilateral.

Very rarely a cerebral cortical lesion in the laryngeal center may cause pseudo-bulbar paralysis. Since the two centers are compensatory, the lesion must be bilateral.

The nerve may be involved at its root or in any part of the trunk,



and such lesions are usually unilateral. The recurrent laryngeal nerve, especially the left, is more apt to be diseased than the superior, on account of its position. Thus, the arch of the aorta is more frequently the seat of an aneurysm than the subclavian; enlarged thoracic glands, neoplasms, and an enlarged thyroid can also damage these nerves. The peripheral filaments may be attacked as part of a multiple neuritis.

In certain cases the muscles become weakened without being paralyzed, this possibly being due to a local neuritis, or to a congestion and inflammation of the mucous membrane from over-use (*clergymen's sore throat*), or as the result of exposure.

The following are the chief forms of paralysis:

(1) *Complete Paralysis*.—By this is generally understood paralysis of all except the crico-thyroid and epiglottidean muscles, though occasionally these may also be involved. Since the cords are paralyzed, phonation is impossible. As a rule, there is no interference with respiration, though the pressure of the in-going air may bring the cords nearer together, and thus produce a certain amount of inspiratory harshness.

As the cords cannot be closed, coughing is impossible, as the air escapes through the glottis, and no expulsive force can be given to it. When the paralysis is unilateral these symptoms will of necessity be modified, and some degree of phonation may be possible. The most common cause of this condition is an involvement of the recurrent laryngeal nerve; the lesion may, however, be nuclear or in the course of the nerve-trunk.

(2) *Paralysis of the Abductors*.—The only special abductor muscles are the posterior crico-arytenoids. When they are involved the glottis fails to open in inspiration, and the unopposed adductors bring the vocal cords together. They are still more closely approximated during inspiration by the column of air, and hence the prolonged, stridulous inspiratory sound. Phonation and expiration are practically unchanged. It is quite likely that many cases supposed to be instances of hysteric spasm of the glottis are really cases of abductor paralysis.

In unilateral paralysis the normal movements of the unaffected vocal cord prevent any marked degree of dyspnea and stridor: phonation is usually hoarse and of a low pitch. In cases of long duration the symptoms become more marked as the unopposed adductors undergo secondary contracture and still further narrow the glottis.

This condition may be due either to central disease or to some local change. The abductor muscles may be degenerated, while all the other laryngeal muscles are healthy, or one or both recurrent nerves may be affected. These nerves innervate both the abductors and adductors, and it is not clearly understood why the abductors alone should suffer when the parent nerve-trunk is involved. At any time it might be a very grave condition, for should any swelling of the cords supervene nothing but a prompt laryngotomy could prevent suffocation.

(3) *Adductor Paralysis*.—The cords move normally during respiration, and hence there is no stridor; as they cannot be approximated, however, phonation is impossible. This condition is met with in hysteria, producing hysteric aphonia, in public speakers who overtax their voices, and also in laryngitis.

The following table, from Gowers' text-book on *Diseases of the Nervous System*, enables one to get a comprehensive idea of the subject:

SYMPTOMS.	SIGNS.	LESIONS.
No voice; no cough; stridor only on deep inspiration.	Both cords moderately abducted and motionless.	Total bilateral palsy.
Voice low-pitched and hoarse; no cough; stridor absent or slight on deep breathing.	One cord moderately abducted and motionless, the other moving freely, and even beyond the middle line in phonation.	Total unilateral palsy.
Voice little changed; cough normal: inspiration difficult and long, with loud stridor.	Both cords near together, and, during inspiration, not separated, but even drawn nearer together.	Total abductor palsy.
Symptoms inconclusive; little affection of voice or cough.	One cord near the middle line, not moving during inspiration; the other normal.	Unilateral abductor palsy.
No voice; perfect cough; no stridor or dyspnea.	Cords normal in position, and moving normally in respiration, but not brought together on an attempt at phonation.	Adductor palsy.

Sensory disturbances of the larynx are rare, and especially hyperesthesia. Anesthesia may be due to hysteria, or to bulbar paralysis, or to disease of the superior laryngeal nerve. It is dangerous, as food may enter the windpipe.

(c) **Cardiac Branches.**—These with branches from the sympathetic form the cardiac plexus. The vagus contains both accelerator and inhibitory fibers, but the latter predominate; therefore irritation of the nerve, either centric or peripheral, will slow the heart's action. Czermak was able to slow the action of his heart by pressing a small tumor in his neck against the vagus nerve. When the function of the nerve is lowered, inhibition is removed and the heart's action becomes rapid. This may be brought about by a toxemic neuritis, by pressure, accidental ligature, or by incised or punctured wounds. Various emotions and nervous states may bring about the same result.

(d) **Pulmonary Branches.**—Both accelerator and inhibitory fibers exist, but in this case the accelerator influence predominates, so that irritation results in increased respiratory movements or even in bronchial spasm, since the bronchial muscles are also supplied by this nerve. It is this nerve that is supposed to be concerned in the production of asthmatic paroxysms. Therefore, when the nerve-function is lowered the respirations become much slower. The nerve is supposed to contain trophic fibers for the lungs.

(e) **Esophageal, (f) Gastric, and (g) Intestinal Branches.**—The esophageal branches are rarely damaged, and irritation (spasm) occurs more frequently than paralysis. The pneumogastric gives the sensory, and in part the motor, nerve-supply to the stomach, and irritation gives rise to increased contractions with some pain.

The sensation of hunger is supposed to be associated with the vagus nerve, and vomiting may result from direct or reflex irritation. Par-

alysis causes some diminution of the gastric contractions. Normally, the vagi accelerate intestinal peristalsis.

**Treatment.**—It is almost always impossible to remove the cause of the above conditions. Syphilitic lesions are probably the most amenable, and in the various laryngeal palsies electricity may be employed, though it is of somewhat doubtful utility, and in abductor palsy may possibly exert a harmful influence by stimulating the adductors. Strychnin and general tonics should be administered. Massage of the larynx may be tried, and in spasmodic conditions attention should be directed to the general physical state. All sources of nerve-irritation should be removed if possible, and bromids, or even chloral, should be given.

#### DISEASES OF THE SPINAL ACCESSORY NERVE.

This nerve consists of two parts—an external or spinal, and an internal or accessory, portion. The latter has already been described in connection with the pneumogastric nerve. It forms the motor portion of that nerve, and is distributed to the laryngeal and pharyngeal muscles. The spinal element arises from the multipolar ganglion-cells in the anterior gray horns of the cervical cord, ascends and enters the cranium through the foramen magnum, and leaves it, after joining with the accessory part, through the jugular foramen. It supplies the sternomastoid muscles and in part the trapezius.

Injury or disease of the nerve may result in spasm or paralysis. Only the spinal part is considered in this section.

#### TORTICOLLIS.

(*Wry-neck.*)

This may be a congenital or an acquired condition.

Congenital torticollis, or “fixed wry-neck,” is the result of an atrophy and shortening of the sterno-mastoid muscle, brought about by some intra-uterine condition or, possibly, by an injury at birth. The right muscle is most commonly affected. The head turns slightly toward the sound side; the eye may deviate, and curvature of the cervical spine may develop.

Facial asymmetry is a usual concomitant of this condition. The face on the same side as the lesion develops less rapidly than the other side, and in time secondary contracture of the unopposed muscles takes place. The torticollis can be cured by tenotomy, but the facial asymmetry persists. Fixation is necessary for a while when contracture exists.

Spasmodic wry-neck may be tonic or clonic. These forms may co-exist, alternate, or occur independently in different individuals. The condition is met with almost exclusively in adults, and occurs most frequently in middle-aged men.

**Pathology.**—No macroscopic or microscopic evidence of any lesion has been discovered, and the condition is probably dependent upon an over-activity of the neurons in the various centers that control the muscles of the affected part.

**Etiology.**—The influence of sex and age has been mentioned; a



neurotic heredity may also predispose. Torticollis may follow habit-spasm, or some injury to the head or neck, or exposure to cold, the latter constituting the "rheumatic" type. In a case of my own, a man of 23, it followed an attack of acute articular rheumatism and was associated with high arterial tension. Rarely, robust, healthy-looking individuals are attacked without any apparent cause. Cervical caries may cause rigidity of the neck, simulating torticollis. The spasm is usually tonic in such cases, as it is in those of the "inflammatory" type, where, in children particularly, enlarged and painful glands are found under the sterno-mastoid.

**Symptoms.**—The occiput is drawn toward the shoulder of the affected side, the chin is elevated, and the face rotated more or less toward the sound side. The sterno-mastoid may alone be affected, or the trapezius may also be involved. In the latter case greater depression of the head takes place. Spinal curvature may ensue, the convexity being toward the sound side. This only takes place in cases that have existed for some time. Clonic spasm is infinitely more distressing and more apt to be permanent.

Some pain and muscular twitching may precede the onset of the attack, though, as a rule, muscular contractions are the first indication. These are mild at first, and rarely abruptly, more commonly slowly, they increase in severity. As the case progresses other muscles, and even those of the arm, become involved. Cases have been described in which certain muscles or groups of muscles in the hand or arm have been primarily affected, the condition gradually spreading from them. The spasm usually ceases during sleep. An attack may cause pain, but, as a rule, it induces merely a feeling of fatigue in the muscles; it is worse if the patient is excited or emotional. Bilateral spasm may occur, the muscles of both sides being equally affected (*retro-collis spasm*). Gowers speaks of a case in which the backward displacement of the head was so great that the face was horizontal and looked directly upward.

**Diagnosis.**—As a rule this is not difficult. When spasm is induced by enlarged and painful glands beneath the sterno-mastoid the age of the patient will be of value in determining the true condition. This usually occurs in children; true wry-neck, on the other hand, very rarely commences before the thirtieth year. Hysteric spasm may also simulate spasmodic torticollis, but it generally occurs in young women, and usually other evidences of hysteria are also present. The *rheumatic type* and the rigidity induced by *caries of the spine* must be differentiated from one another and from spasmodic wry-neck. If the rigidity comes on suddenly, following exposure to cold or wet, and the pain is not increased at night or by depressing the head upon the spine, and is relieved by hot applications, the condition is probably rheumatic. When the rigidity and pain are of slow onset, without history of exposure, and the pain is both worse at night and is increased by depressing the head upon the spine, but is relieved by elevating the head, the condition is very probably one of caries of the spine.

**Prognosis.**—Very rarely the torticollis may diminish or even cease after an existence of months or years. Usually, however, it is persistent, either being stationary or slowly increasing in severity and widening in range. The prognosis must always be guarded, and in severe cases grave as to recovery, though the disease does not shorten life.

**Treatment.**—Generally very little can be expected from medication. Bromids, morphin, chloral, hyoscyamus, or cannabis indica may be tried, as may the various forms of counter-irritation. Morphin administered hypodermically, has been most effectual in some cases, but the danger of establishing the habit should not be forgotten. Galvanism should be tried, the negative pole being placed over the occipital region and the positive over the affected muscles. Nerve-stretching and tenotomy of the affected muscles is of very little value. The only surgical procedure that has proved of any distinct value is neurectomy, with excision of a part of the nerve to prevent reunion. This necessarily causes paralysis and atrophy of the muscles supplied; but, since it often abolishes the spasm, the slight loss of power and the interference with the movement of the head are comparatively infinitesimal. The results, however, are not uniform even so far as the spasm is concerned.

#### PARALYSIS OF THE SPINAL ACCESSORY NERVE.

The accessory portion has been previously considered in describing the laryngeal branches of the pneumogastric.

In the spinal portion the nuclei may be involved in degenerative lesions of the motor region of the spinal gray matter. The nerve-trunk may be damaged by pressure from exudative products (meningitis), tumors, or caries, with resulting paralysis and wasting of the sternomastoid and, in part, of the trapezius. This latter muscle is also supplied by the cervical nerves. The patient has difficulty in rotating the head to the side opposite that on which the paralysis exists, and the affected muscle does not stand out in movements of the head. Unless secondary contraction of the unopposed muscle sets in, no deviation occurs when the head is at rest. The only portion of the trapezius that is involved in paralysis of the external part of the eleventh nerve stretches from the occipital bone to the acromion. The normal contour of the neck is lost in such cases, and the ability to raise the arm is interfered with because the trapezius cannot fix the scapula, the fulcrum of the deltoid. Bilateral paralysis may occur as in progressive muscular atrophy; if both sternomastoids are involved, the head falls backward; if both trapezii, it falls forward.

The **treatment** is that of the underlying cause. If the lesion is nuclear, practically nothing can be done. If the condition is due to pressure, in some cases relief may be obtained. Electricity and massage should be employed during the recovery of the nerve.

#### DISEASES OF THE HYPOGLOSSAL NERVE.

The nucleus of the twelfth cranial nerve is in the most posterior portion of the floor of the fourth ventricle. It is said by some observers that the nuclei of the fibers for the palate and vocal cords that run in the spinal accessory nerve may be in the lower part of the twelfth-nerve nucleus.

The cortical center for this nerve is in the lower part of the ascending frontal convolution, in the neighborhood of the cortical facial center. This propinquity probably explains the simultaneous involvement

of the facial and lingual muscles in some cases. The hypoglossal is the motor nerve for the tongue and for most of the muscles attached to the hyoid bone. Spasm or paralysis may follow disease of the nerve.

**Spasm** may be either unilateral or bilateral. It is probably met with most commonly in hysteria, or as a part of some general convulsive condition, as epilepsy or chorea. It may also be associated with facial spasm, as mentioned above. Irritation of the fifth nerve (dental caries, ulceration of the gums) seems to be responsible for some cases. "Paroxysmal clonic spasm" is a form in which the tongue is rapidly thrust in and out. Various sensations in the affected region may precede the attack. A rare form—*aphthongia*—is induced when an attempt to speak is made. The prognosis in this condition is good, and a general tonic treatment is indicated.

**Paralysis** may result from supra-nuclear, nuclear, or infra-nuclear lesions.

*Supra-nuclear*.—The lesion may be anywhere between the cortex (lower part of the ascending frontal gyrus) and the medulla, and causes paralysis on the opposite side. In this condition the affected muscles do not atrophy nor do they show any electric change.

*Nuclear*.—The lesion is usually degenerative. It may either be of sudden onset (vascular), less rapid, but still acute (inflammatory), or it may be chronic, as in bulbar palsy or tabes dorsalis. The nuclei are so close together that the condition is almost invariably bilateral.

*Infra-nuclear*.—The fibers may be injured by the pressure of neoplasms or by the products of meningitis or of syphilis. Disease of the bone may also involve the nerve in its passage through the foramen. More rarely, some traumatism or disease of the upper cervical vertebrae may simultaneously injure the eleventh and twelfth nerves.

**Symptoms**.—Paralysis and atrophy of one or both sides of the tongue and fibrillary twitchings may be noted, and if the condition be unilateral, the tongue when protruded deviates toward the affected side. Articulation, mastication, and swallowing are but very slightly interfered with. In the bilateral form, however, these are very much impaired; the tongue cannot be protruded and lies motionless on the floor of the mouth. The atrophy is muscular. This throws the mucous membrane into deep folds. Sensation and taste are unaltered.

**Diagnosis**.—If the lesion is supra-nuclear, there is hemiplegia on the same side as the lingual paralysis, without atrophy of the tongue-muscles. When nuclear, it is, as has been said, generally bilateral and forms part of a bulbar paralysis. There is also wasting of the lingual muscles. When the fibers are involved in the medulla, there is paralysis of the tongue on one side, of the limbs on the other, and the tongue deviates from the paralyzed side of the body. Outside the medulla the condition is, as a rule, unilateral, and the spinal accessory fibers are frequently involved. In the nuclear and infra-nuclear varieties there is wasting of the muscles.

The **prognosis** is usually unfavorable, and the **treatment** consists of a course of general tonics and of mercury and the iodids, with counter-irritation. Electricity may also be tried.



## DISEASES OF THE SPINAL NERVES.

## DISEASES OF THE CERVICAL PLEXUS.

**Phrenic Nerve.**—This nerve is usually involved as a result of some lesion of the ganglion-cells in the anterior gray horns at the level of the third or fourth cervical nerve. The trunk may be damaged by pressure, as by aneurysm or neoplasms, or by traumatism, or it may be the seat of neuritis. More or less immobility of the diaphragm follows, amounting in some cases to complete paralysis. This is not readily seen with the patient at rest, and in women it is specially hard to observe, as their breathing is chiefly of the costal type. The abdomen moves in in inspiration, and out in expiration, forming the reverse of the normal movements. Immobility of the diaphragm may also occur in peritonitis, diaphragmatic pleurisy, and in large pleural effusions. Exertion readily causes dyspnea, and pulmonary diseases are apt to be exaggerated as the products of secretion accumulate. This is most apt to occur when the condition is bilateral, as it usually is in the presence of cord-lesions. Other muscles always suffer in this form in addition to the diaphragm. When the nerve alone is involved the affection is generally unilateral.

## DISEASES OF THE BRACHIAL PLEXUS.

This may either be involved *in toto*, or any of its branches may be affected separately, or the nerve-roots that unite to form the brachial plexus.

Considering first the roots, the only nerve worthy of notice arising from them is the posterior thoracic, which supplies the serratus magnus muscle. This may be injured directly by pressure, as in the carrying of heavy loads on the shoulder or by a fall or other traumatism. Rarely, it follows exposure to cold. Its involvement may be a part of an anterior polio-myelitis or of progressive muscular atrophy. When the muscle is paralyzed the posterior edge of the scapula stands out prominently, and particularly when the arm is moved forward. Neuralgic pains in the neck generally precede the neuritis. The course of the disease is always slow. During the early stage counter-irritation, the iodids and mercury internally, and later electric stimulation to keep up the tone of the muscles, constitute the treatment.

**Combined Paralysis.**—Two or more nerves, or even the entire plexus, may be involved at one time by new growths in the cervical region, neuritis, stretching or rupture of the nerves by wounds, fractures, or dislocations, and particularly by subcoracoid dislocation. Duchenne has described a form of palsy produced in infants during birth by pressure due to some malposition or to injury by the finger or a hook. Brachial neuritis may follow some injury to one of the nerve-branches (ascending neuritis), or it may be primary. The latter variety is rare, and usually occurs after middle life, especially in cases with a gouty history. Paroxysmal or continuous pain, increased by any movement of the arm and tenderness on pressure over the affected nerves, is the chief symptom. If on the left side, it simulates angina pectoris.

**Individual Nerves of the Arm.**—These may be damaged by pressure

due to a tumor, an aneurysm, or to callus. *Sleep-palsy* and *crutch-palsy* are both pressure-palsies. The nerves may also be contused or torn in fractures or dislocations, and palsy may follow a fall or blow upon the shoulder; I have seen it occur in a heavy man after a fall upon the hand. Primary or secondary neuritis may develop, and, very rarely, neuromata appear.

The *supra-scapular* nerve supplies the supra- and infra-spinati muscles. Paralysis causes imperfect outward rotation of the humerus and rotation of the scapula, with elevation and inversion of the lower angle. Various movements of the arm are thereby interfered with, and the limb tires very readily. More work is thrown on the deltoid, and in time it hypertrophies, causing it to stand out more prominently against the infra-spinatus. The skin over the scapula is usually anesthetic.

The *circumflex* nerve supplies the deltoid and teres minor and the skin over the deltoid and the shoulder-joint. Paralysis results in inability to raise the arm and in wasting of the muscles, with or without anesthesia. Adhesions may form in the joint.

The *musculo-spiral* nerve is more often paralyzed than any other nerve of the arm, its position rendering it particularly liable to pressure. It supplies the triceps and supinator muscles, and is the extensor nerve of the arm. It also supplies the skin on the radial side of the dorsal surface of the hand, the back of the thumb, and the index and radial side of the middle finger. A lesion high up results in paralysis of the extensors of the elbow, wrist, and hand, and of the supinators. Probably the point most commonly attacked is about the middle of the humerus. In such cases the triceps escapes. The characteristic symptoms, however, are wrist-drop and finger-drop, consisting of an inability to extend the hand on the forearm, also the first phalanges of the fingers and thumb. In pressure-palsies the power of supination is usually lost also. Sensory symptoms vary, and are seldom pronounced. There may be slight impairment or tingling or burning sensations.

This condition can usually be differentiated from lead-palsy by the rapidity of onset—by the fact that pressure-palsies are almost invariably unilateral, and that the supinators are involved. Lead-palsy has a slow onset and is bilateral, generally without supinator involvement. Loss of sensation precedes the pressure-palsy. The history too will generally throw some light on the case. I have seen a case of right-sided unilateral wrist-drop in a man who worked in lead with his right hand only. Bilateral wrist-drop may occur in any form of toxic neuritis, but the involvement of other nerves, the manner of attack, and the history of the case will serve to simplify the diagnosis.

Recovery follows in almost all cases of musculo-spiral nerve-involvement, though in cases in which qualitative nerve-changes have taken place it is necessarily delayed.

The *treatment* is that of neuritis.

The *median nerve* supplies the pronators, digital flexors, except the ulnar half of the deep flexor, the radial flexor of the wrist, the abductor and flexor muscles of the thumb, and the two radial lumbricales. It furnishes sensation to the radial side of the palm and front of the thumb, and to the front and back of the first and second and half of the third fingers. This nerve may be the seat of an injury or of neuritis, but is

seldom involved alone. The most striking symptoms are wasting of the thenar eminence and an inability to oppose the thumb to the tips of the fingers. Further pronation is only possible in so far as the supinator longus subserves that function—viz. the misposition. Ulnar flexion of the wrist alone remains. Flexion of the phalanges is interfered with. Sensation may or may not be lost.

The *ulnar nerve* supplies the ulnar flexor of the wrist, the ulnar half of the deep flexor of the fingers, the muscles of the little finger, the adductor and inner head of the short flexor of the thumb, the interossei, and some of the lumbricales. It supplies with sensation the front of one and a half and the back of two and a half fingers on the ulnar side. Paralysis causes radial deviation of the hand in flexion of the wrist, loss of adduction of the thumb, and inability to move the little finger. The hypothenar prominence disappears.

The first phalanges cannot be flexed, and the second and third cannot be extended. This is exaggerated in old cases, though still it is not so marked as the "claw hand" of progressive muscular atrophy, since the first two lumbricales escape, being supplied by the median nerve. Sensory symptoms vary.

The *diagnosis* is usually easy. It is well to remember that, since this nerve is the lowest in its point of origin of any considered in this group, ascending cord-diseases will involve it before any of the other brachial nerves. It may also be damaged by disease limited to the lowest part of the cervical enlargement of the cord.

#### DISEASES OF THE LUMBAR AND SACRAL PLEXUSES.

The **lumbar plexus or its branches** may be involved by abdominal growths, enlarged glands, psoas abscess, disease of the vertebræ, neuritis, and rarely by wounds or dislocation of the hip or during parturition.

*The Obturator Nerve.*—When the power of adduction of the thigh is lost and the affected leg cannot be crossed over the other, outward rotation is somewhat impaired.

*Anterior crural nerve* paralysis causes loss of power and wasting of the extensors of the knee, loss of knee-jerk, and anesthesia of most of the thigh and the inner side of the leg and foot.

The *superior gluteal nerve* supplies the gluteus minimus and medius muscles. When it is involved adduction and circumduction of the thigh are lost.

The **sacral plexus and its branches** may be damaged by pelvic neoplasms or inflammation, neuritis (generally secondary to sciatic nerve-involvement), pressure during labor, wounds, dislocations, aneurysms, and diseases of the bone.

The *small sciatic nerve* supplies the gluteus maximus muscle. It is seldom involved alone. Lesions cause difficulty in rising from the sitting posture and anesthesia of the back of the thigh and of the upper part of the leg posteriorly.

The *great sciatic nerve* supplies the flexors of the leg and the muscles below the knee, and also sensation to the outer half of the leg, the sole, and part of the dorsum of the foot. Paralysis causes more or less



interference with the act of walking, anesthesia in the part supplied, and wasting of the muscles.

The *external popliteal* or *peroneal nerve* supplies the *tibialis anticus*, the *peronei*, the long extensor of the toes, and the *extensor brevis digitorum*; it also supplies sensation to the outer half of the front of the leg and to the dorsum of the foot. Paralysis causes foot-drop and toe-drop, rendering it necessary to lift the leg high in walking, so that the foot will clear the ground; this constitutes the *steppage gait* referred to in the section on Neuritis. The region supplied is anesthetic.

The *internal popliteal nerve* supplies the *popliteus*, *tibialis posticus*, the calf-muscles, the long flexors of the toes, and the muscles of the sole. When paralyzed, flexion of the foot and toes is impossible, and sensation is lost over the back of the leg in its lower part and over the sole. In old cases talipes calcaneus results. The plantar nerves are rarely, if ever, involved alone.

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## ACUTE ASCENDING PARALYSIS.

(*Landry's Paralysis.*)

**Definition.**—An acute paralysis, beginning in the legs and ascending by way of the trunk and upper extremities, and ultimately involving the *medullary* centers. It usually runs a short course, and, as a rule, terminates in death.

**Pathology.**—Although in many cases neither gross nor microscopic lesions have been found, either in the cells, peripheral fibers, or muscles, it is believed to be either an acute myelitis or an acute polyneuritis, the weight of opinion seeming to favor the latter view. Ross arrived at the latter conclusion after an analysis of 93 cases. Nauwerck, Barth, and Centanni hold the same belief, and the latter has discovered a bacillus in the lymph-spaces of peripheral nerves. Remlinger<sup>1</sup> has reported a case occurring in a young man in whom paraplegia developed acutely, and eleven days later death resulted from bulbar involvement. Postmortem the cord was found congested in the region of the anterior horns. Microscopic examination revealed the presence of inflammation in the cervical cord. The multipolar ganglion-cells of the anterior horns were degenerated, and between them were found streptococci. Pure cultures of streptococci were obtained from the cord at various levels, but they were non-pathogenic for the rabbit. R. and F. Schultze and Sinkler have also reported cases in which the only postmortem lesion was myelitis, yet, as stated, the majority of observers believe it to be a neuritis. That it is primarily due to some toxemia, however, as originally claimed by Westphal, cannot be gainsaid. The prodromes, when present, are suggestive, and the enlargement of the spleen, which is a constant concomitant, and more rarely the lymphatic enlargement and albuminuria, are all confirmatory. That the poison should have a selective tendency, since the nervous involvement is chiefly or solely motor, is not unique. We meet with toxic paralysis of the motor muscles of the eye, also with lead-palsy.

<sup>1</sup> *Gazette hebdomadaire de Médecine et de Chirurgie*, No. 27, 1896.

**Etiology.**—No definite cause is known. It has followed cold and exposure, traumatism, and the infectious fevers, including influenza. Remlinger's case, quoted above, followed malaria. It occurs in males chiefly between twenty and forty years.

**Symptoms.**—In the most acute cases there are practically no prodromal symptoms other than malaise and possibly chilly sensations. Weakness, followed in a few hours or a day or two by paralysis, develops in the lower extremities. One may be involved a few hours earlier than the other. It spreads toward, and soon involves, the trunk also, and in quick succession the arms. The third and usually fatal stage is reached when bulbar symptoms develop. Very rarely the upper extremities may be first attacked. Death may occur in forty-eight hours. The paralysis is a flaccid one; the muscles can be passively moved without offering any resistance. Wasting sets in, but no electric changes. In less acute cases a decided febrile stage precedes the onset of paralysis, chills, fever, malaise, and possibly formication or even sharp pain. In any case the later symptoms are pre-eminently or solely motor. Sensory symptoms when present are very slight. Sensation may be delayed, and the reflexes are generally absent; accordingly, there is edema or sweating. The bladder and rectum are not implicated, nor do bed-sores develop. As stated, when the bulb is attacked death generally follows, due to cardiac or respiratory failure or to interference with deglutition. There are no cerebral symptoms.

**Course.**—Death may occur in from forty-eight hours to a few weeks. A few cases of recovery have been reported, however, in some of which paralysis had been widespread, even reaching the bulb, judging from the labored respiration. When improvement takes place, it does so in the reverse order to the onset, so that the part last affected is the first to recover. It is much slower than the invasion.

**Diagnosis.**—The rapid onset of a paralysis that usually ascends, the relaxation of the muscles, slight wasting, if any, and the absence of electric changes and of sensory symptoms, with or without fever, serve to make the diagnosis, and to distinguish Landry's disease from poliomyelitis, neuritis, and spinal hemorrhage. For the differential diagnosis between Landry's paralysis and acute myelitis, see page 1072.

**Prognosis.**—Always grave, particularly if bulbar symptoms occur, and especially if they appear early.

The **treatment** is essentially the same as that for any acute disease of the cord or nerves—*i. e.* rest, freedom from all excitement or worry, moderate purgation and diaphoresis; ergot, belladonna, and iodids internally. Should the patient survive, electricity and massage should be administered.

## II. DISEASES OF THE SPINAL CORD AND ITS MENINGES.

### DISEASES OF THE MENINGES.

**MENINGITIS** is very rarely a primary condition. Both the dura and pia may be involved. In the former case the inflammation is usually due to some morbid condition of the vertebræ, while in the latter it is secondary to some toxemia, as in pyemia, sepsis, pneumonia, typhoid, or the acute exanthemata. It may be part of a tuberculous condition (*vide* Tuberculosis, p. 278) or of epidemic cerebro-spinal meningitis. Injuries and, it is said, exposure to cold, also lead to inflammation of the meninges of the cord.

#### PACHYMENINGITIS.

**Definition.**—Inflammation of the dura mater. The dura may be involved on its outer or inner surface (*pachymeningitis externa* or *interna*), or the loose connective tissue between the dura and bony canal may be the seat of a peripachymeningitis.

**Pachymeningitis externa** is always secondary, and usually results from syphilitic or carious affections of the bone, or from pressure due to tumors or to traumatism. It may either be acute or chronic. Of the latter type, those cases due to Pott's disease are most common. The membrane is involved to a greater or less extent. The internal surface may escape entirely, or it may be slightly roughened and adherent to the arachnoid; externally, however, the dura is usually thickened, rough, and covered with a cheesy material.

**Pachymeningitis interna** was first described by Charcot in 1871, and named "*pachymeningitis cervicalis hypertrophica*." It is of obscure origin. The dura is generally much thickened, and gives the impression of being made up of a number of concentric layers. The pia is only involved to a slight degree as a rule. Areas of degeneration may occur in the cord, as may also dilatation of its central canal. As implied by the name, this variety of pachymeningitis is found chiefly in the cervical region, and the clinical symptoms result from involvement of the nerve-roots. It is a chronic process, and has been divided into three periods, as follows: (a) *The painful period*, lasting, as a rule, two or three months, in which severe neuralgic pains exist, their location being determined by the roots involved. They are mostly in the occiput and upper extremities, however. Early there may be hyperesthesia, numbness, tingling, and, rarely, an herpetic eruption. (b) *The Paralytic Period*.—As a result of compression of the motor roots an atrophic paralysis of the upper extremities develops. A peculiar selective tendency is manifested, the radial nerve being spared, while the median and ulnar nerves are involved. This results in a modified "claw-hand" deformity and in an over-extension of the wrists, with flexion of the fingers. Anesthesia may be noted. (c) *Spastic Paraplegia*.—This results when the compression has produced degeneration of the cord. Generally, there are paresis of the lower extremities and increased reflexes, but no muscular wasting, since the trophic centers are intact. Occasionally, however, anesthesia



and paralysis of the legs and bladder develop, bed-sores following, with death from exhaustion.

The *prognosis* must be guarded, each case being carefully looked into and diagnosed from amyotrophic lateral sclerosis, syringomyelia, and from pressure by tumors. From the latter the condition is very difficult to differentiate, cervical spondylitis and neoplasms often giving rise to the same symptoms. The first-named condition does not give rise to sensory disturbances; moreover, bulbar symptoms are often present, the lower extremities atrophy, and the bladder functions are preserved. Syringomyelia induces characteristic changes in thermic sensibility, and often anesthesia, but rarely severe neuralgic or radiating pains.

**Pachymeningitis hæmorrhagica interna**, or *hematoma of the dura mater*, may occur in any part of the cord, and is usually associated with a similar condition in the cerebral dura. Cysts may be found in the inner surface of the dura, containing broken-down blood-cells and hematoïdin crystals, and in their neighborhood an increase of fibrous tissue may be noted. The condition occurs most frequently in alcoholics or general paralytics.

*Treatment* is not of much avail. Counter-irritation, potassium iodid, and electricity are the chief measures.

#### LEPTOMENINGITIS.

**Definition.**—Inflammation of the pia mater. This may be either acute or chronic.

#### ACUTE LEPTOMENINGITIS.

(*Acute Spinal Meningitis.*)

**Pathology.**—The vessels are injected, the membrane becomes cloudy, a sero-fibrinous or purulent exudate either surrounds the cord or may only exist in patches, and in the more severe cases the cord itself is involved (*meningomyelitis*). The spinal meninges alone may be involved to a greater or less extent, but as a rule, the cerebral meninges are similarly involved.

**Etiology.**—Rarely is this a primary disease. It may be met with—(1) In tuberculosis, in which the cerebral symptoms predominate. (2) In cerebro-spinal meningitis, an epidemic, specific infectious disease. (3) As a condition secondary to one of the infectious fevers, as pneumonia, typhoid, and influenza. This, however, is very rare. It should be remembered that many cases presenting clinically the picture of meningitis show absolutely no *postmortem* lesions of the cerebral or spinal membranes. This is especially true of pneumonia and influenza. The condition in such cases is probably a toxic encephalopathy. (4) In myelitis. In certain cases the pia becomes involved, due to extension from the cord. (5) In injuries. (6) As a result of cold and exposure, though probably rarely.

**Symptoms.**—These are chiefly pain in the back, often excruciating, with fixation, retraction of the head, tenderness on pressure along the spine, tremors or spasm of the muscles, and various sensory disturbances. Reflexes are early increased, and later diminished or absent. Should the cord be involved, paralysis, incontinence of urine and feces, and even bed-sores, may develop. The symptoms are more fully discussed in speaking of the tuberculous and epidemic varieties.

**Diagnosis.**—It is often very difficult to differentiate the several

varieties of spinal meningitis, and equally so to decide whether the case is actually meningeal when some other disease is present. Even bulbar symptoms may be present without postmortem lesions; I have seen this typified in a case of Bright's disease. The tuberculous form is readily diagnosticated, especially if any collateral evidence of tuberculosis exists. It is a point of some value in the diagnosis to note the absence of marked leukocytosis in tuberculous and its presence in purulent meningitis. The presence of Kering's sign is in favor of *cerebro-spinal meningitis*.

*Spinal paracentesis* or *lumbar puncture*, first introduced by Quinke of Kiel in 1891, is a most valuable diagnostic measure and simple of application. He was first led to adopt it by the knowledge that a free communication exists between the subarachnoid spaces of the brain and spinal cord through the foramen of Magendie; hence he conceived the idea of a lumbar puncture supplanting the older method of tapping the lateral ventricles in cases of hydrocephalus. Later, he used it in meningitis. Therapeutically, it is of little value. The patient should be in a sitting posture with a slight forward inclination of the trunk. The puncture is then made between the third and fourth lumbar vertebrae and a little to one side of the middle line. Absolute cleanliness should be observed, and the needle introduced slowly until the fluid begins to flow by its own pressure.

The **prognosis** is unfavorable as a rule, particularly in the tuberculous form.

The **treatment** is the same as that of cerebro-spinal meningitis (*vide* p. 131).

#### CHRONIC LEPTOMENINGITIS.

This disease may follow the acute form or be due to chronic alcoholism, syphilis, trauma, or disease of the cord.

*Pathology.*—The pia is cloudy and swollen, and often adherent to the arachnoid, or all three membranes may be glued together. They are usually injected. An exudate fills the meshes of the arachnoid. The periphery of the cord is occasionally affected at the same time.

*Symptoms.*—These are not well marked. Unless the nerve-roots are involved the symptoms are slight or none at all exist; however, pains of a radiating character, stiffness, tremors, hyperesthesia, herpes, and even paralyses, may occur. The course is slow, and may extend over many years.

The *prognosis* is unfavorable ultimately.

The *treatment* consists in the use of iodids and mercury internally, and the application of baths, and counter-irritation along the spine.

#### HEMORRHAGE INTO THE SPINAL MENINGES.

(*Meningeal Apoplexy; Hemorrhachis.*)

(a) **Extrameningeal hemorrhage** occurs when the blood is between the dura and spinal canal.

(b) **Intrameningeal hemorrhage** is that in which the bleeding takes place within the dura.

Large hemorrhages are very rare in any case, but are more common in the extrameningeal form; they result from trauma or rupture of an aneurysm. The peridural space will accommodate a large amount of blood without giving rise to pressure-symptoms. Caries of the vertebræ or carcinoma may cause hemorrhage by erosion and rupture of a blood-vessel. The intra-meningeal form is somewhat more common, and may either result from meningitis or occur as a complication of any of the infectious diseases. In such cases the hemorrhages are small and scattered. It may also occur in convulsive disorders or in strychnin-poisoning. Rupture of an aneurysm at the base of the brain may give rise to extensive hemorrhage, and in a case of syphilitic ventricular apoplexy in a young man I found, *postmortem*, that the blood had leaked out and infiltrated the spinal meninges for some distance.

**Symptoms.**—When the hemorrhage is large enough to cause pressure, the symptoms are very acute, apoplectiform indeed, but consciousness is preserved. Generally, however, they are quite indefinite. In any case they depend upon the degree and location of the compression. At first they are irritative—viz. hyperesthesia, paresthesia, neuralgic pains that are radiating in character, herpes, muscular irritability, tremors, or contractions. Later, paralytic symptoms may develop, as anesthesia and bladder- and bowel-symptoms, girdle pains, or, when the lesion is high up, interference with respiration, and pupillary changes.

The **diagnosis** is often difficult, unless the onset is sudden and explosive.

The **prognosis** depends on the cause and extent of the hemorrhage. If small in amount, absorption is usually prompt, with little or no disturbance of function remaining.

The **treatment** consists of rest, ice to the spine, counter-irritation, wet- or dry-cupping, leeches or venesection, ergot, opium or gallic acid internally, and later the iodids and electricity. In certain cases operative procedures, with a view to removing the clot, may be justifiable.

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## DISTURBANCES OF CIRCULATION IN THE CORD.

THESE include qualitative and quantitative changes in the blood, and morbid conditions of the vessel-walls.

The blood-vessels may be the seat of peri- or endarteritis, and rarely military aneurysms may develop. Embolism and thrombosis also occur, the former much less frequently than the latter, which is prone to follow sclerotic changes in the vessels, giving rise to ischemia and ultimately to softening.

**Congestion.**—We are justified in noticing this as a possible cord-lesion, but it is questionable if it has any clinical significance. It is safe to assume that it occurs in the general stasis of circulatory disorders, yet no characteristic symptoms develop. It is very rarely met with *postmortem*.

**Anemia.**—This condition, like the preceding, rarely gives rise to symptoms. Dr. William A. Hammond has described a certain group of symptoms as due to spinal congestion, and another to spinal anemia,



but his teachings on this point are not generally accepted. Simple anemia of the cord, *per se*, cannot be recognized clinically. During the past few years, however, many observers have reported certain and distinct *post-mortem* findings, with or without clinical evidences that the same have occurred during life, in cases of grave anemia, particularly in pernicious anemia and to a lesser extent in leukemia. Whether the anemia is the direct cause of the cord-lesions, or, what seems more likely, whether the anemia and cord-lesions are both produced by some toxemia, remains to be proved. Lichtheim was the first to recognize and call attention to the subject, although Leichtenstern in 1884 reported 2 cases of "tabes" associated with anemia. He was unable to elicit any history of syphilis, nor had crises occurred. In 1887, Lichtheim reported 3 cases of pernicious anemia that presented at the same time symptoms pointing to a lesion of the cord. The first had weakness, ataxia and rigidity of the legs, lowered knee-jerks, paresthesiæ, and normal pupillary reaction. The second case was similar, but the third had lancinating pains and absent knee-jerks. Autopsies were made upon the first two. More or less complete degeneration of Goll's columns was found, and the pyramidal tracts were also involved, but to a lesser extent. Small foci of degeneration were also found in the anterior and lateral columns. He regarded it as due to a toxic process. Later, his pupil Minnick published several cases in which no evidence of spinal-cord disease occurred *intra vitam*, yet in none of them was a normal cord found *postmortem*. Some showed the same changes that occur in hemorrhages of the cord, while others presented degenerative changes of varying degrees in the posterior columns. In all of them Clarke's and Lissauer's columns and the posterior roots were normal. Dr. K. Petren, a Swedish physician, has described a case in which Lissauer's column was also involved. He holds the same view as Lichtheim with reference to the cause, and significantly mentions the changes that take place in the nervous system in certain cases of diabetes evidently toxic in nature. Since then Williamson has reported 3 cases of diabetes mellitus in which degeneration of the posterior columns was found. Cord-changes have been found also in leukocythemia, chronic jaundice, and in persons reduced by other long-standing illnesses. Dr. Putnam has published a series of 8 cases belonging to this latter category. They were adults past middle life, the majority being women in an enfeebled condition. *Postmortem* he found system-sclerosis of the spinal cord, associated with diffuse collateral degeneration. He also found some degeneration of the cells of the gray matter, and, to a less extent, of the peripheral nerves. Nonne and Eisenlohr in Germany, Taylor and Bowman in England, and Burr in this country, have also reported cases of cord-lesions associated with grave anemia. The chief symptoms described have been progressive weakness, paresis of all the extremities (particularly the lower), ataxia, and in some cases weakness of the bladder. The knee-jerks are either increased, diminished, or absent. There are sensory disturbances (paresthesia) and lancinating pains occur very rarely. Whatever part the anemia plays, it seems that the fundamental cause is a toxemia, and I venture to say that this fact may throw some light on those cases of tabes, and even of chronic myelitis, in which no history of syphilis or other predisposing cause can be obtained. Further, it seems to me that they may be compared with those toxic conditions described by Duke

in England and McLane Hamilton in this country that have their chief incidence upon the cellular elements of the brain.

**Treatment.**—The indications are to keep the emunctories active. High enemata should be given, flushing the bowel with large amounts of sterile normal salt-solution. Internally, calomel, salol, beta-naphthol, arsenic, and iron may be employed.

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## HEMORRHAGE INTO THE SPINAL CORD.

(*Hematomyelia; Spinal Apoplexy.*)

THIS is a very much less frequent occurrence than cerebral hemorrhage. It is usually due to traumatism, but may follow cold or exposure or some severe strain or over-exertion (in the latter probably only when the vessels are atheromatous). Hemorrhage may occur in cases of myelitis, epidemic cerebro-spinal meningitis, syringomyelia, tumors of the cord, convulsive disorders, and infectious diseases; it is, however, usually small. If the hemorrhage is extensive, disruption of more or less cord-substance necessarily follows. An area may exist large enough to cause distention of the cord without rupture, and from this extravasations may take place in the cord-substance above and below. Unilateral hemorrhage may occur, the gray matter being chiefly involved. If of recent origin, fresh blood will be found *postmortem*; but if of long standing, a brown or brownish-yellow area will be noted, consisting of disintegrated blood-corpuscles, cell-detritus, and hematin crystals.

The *symptoms* necessarily vary according to the region involved. The hemorrhage may be sudden, giving rise to an apoplectic onset; or gradual, with slowly increasing symptoms. There is generally a back-ache, followed by paralysis, a loss of sensation and of the reflexes, and in some cases a loss of control of the bladder and bowel. In less grave cases the early symptoms will be those of irritation, while later paralytic symptoms supervene. If the hemorrhage is slight, absorption soon takes place, with complete recovery; but quite often more or less paralysis remains. Myelitis develops in some cases, the patient growing progressively worse and dying of exhaustion. Dr. C. E. Riggs has reported a rather unique case in a woman forty-five years of age, who developed paraplegia after a nervous shock three years before coming under his observation. When he first saw her she had impaired sensation of the lower limbs and of the trunk as far up as the xiphoid cartilage. The legs were spastic, with increased reflexes. She had neither lancinating pains nor ataxia, but was profoundly anemic, and grew progressively worse until death ensued from exhaustion. *Postmortem*, an area of extravasated blood was found in the mid-dorsal region of the spinal canal, and hardening degeneration was noted in the anterior and crossed pyramidal tracts, direct cerebellar and posterior columns, and in Lissauer's tract. The degeneration extended from the first cervical to the fifth lumbar vertebra. This case was remarkable—first, from the fact that the hemorrhage of the cord was due to anemia; secondly, on account of the extent of the degeneration, and particularly because of the fact that Lissauer's column was involved.

The *diagnosis* is always difficult, for when of sudden onset, unless aided by the etiology, it will be impossible to diagnose the condition from spinal meningeal hemorrhage. In other cases it must be differentiated from myelitis and multiple neuritis.

*Treatment*.—Rest, ice locally, and the internal use of ergot and opium make up the treatment.

## ACUTE MYELITIS.

(*Myelitis; Acute Diffuse Myelitis; Transverse Myelitis; Spinal Malacia.*)

**Definition.**—An inflammation, with softening, of the cord, giving rise to various groups of symptoms depending upon the region or regions involved, and not, therefore, as constant in its symptomatology as the systemic nervous diseases (tabes dorsalis, lateral sclerosis).

**Pathology.**—The cord may present little or no change to the naked eye, or in the most acute cases it may be diffuent. Between these extremes many grades exist in which the pia will be found congested and adherent, the cord being more or less ingested and areas of softening, and even cavities, being found. Three forms of softening are spoken of by some writers—the red, yellow, and gray—depending upon the predominance of blood, fat, or connective tissue respectively. The *postmortem* finding depends upon the duration of the disease; the more chronic the course, the greater the amount of nervous connective tissue (neuroglia), and in consequence sclerosis will be the predominant feature. The nerve-cells and fibers are found in various stages of disintegration, the former being swollen, vacuolated, granular, and their processes broken and in many cases missing; while the latter swell, the myelin breaks up, undergoes fatty change, and is removed, and the axis-cylinders finally break up and disappear. A single area of degeneration may exist centrally, in one half of the cord, transversely, or many localized or widely-disseminated areas may be found; but above and below all of them will be found degenerated fibers—ascending and descending degeneration—due to a solution of continuity between the cell-body and its axis-cylinder process.

**Etiology.**—Myelitis may follow exposure (especially in alcoholics), the infectious fevers (chiefly measles and small-pox), and it may be due to traumatism or disease of the vertebræ (caries, malignant disease). Syphilis is also said to cause it, though it may only act as a predisposing agent. It has also been described as following peripheral neuritis, ascending neuritis, and we meet with some cases in which pregnancy seems to act as the predisposing cause. Embolism and thrombosis may rarely cause it. It is most common in males, generally from fifteen to thirty years of age.

**Symptoms.**—These will vary according to the seat and extent of the lesion. In the most acute form the course of the disease is quite rapid, reminding one of hemorrhage into the cord or membranes; the onset, however, is not so explosive, and, though rapid, it is not sudden. It is



most apt to follow cold or exposure. The most acute case I have ever seen occurred in an alcoholic who had lain out one night in a drunken stupor. There may be chills and fever, malaise, backache, pains in the limbs, and, rarely, convulsions; quite often, however, there is no warning. Motor weakness develops, and is rapidly followed by paralysis. Some irritative sensory symptoms appear, as hyperesthesia and paresthesia, and then more or less complete anesthesia supervenes. The reflexes are generally lost; there is incontinence of urine and feces, and bed-sores and cystitis develop with frightful rapidity. The temperature now rises to 105° F. (40.5° C.) or even higher, and typhoid symptoms, exhaustion, and death close the scene. I have seen 1 case that developed in a woman a few days after delivery and proved fatal in six days.

Acute transverse myelitis is the type most frequently met with, however, the lesion being generally situated in the dorsal cord. The constitutional symptoms marking the onset are more pronounced than in the previous type and are of longer duration; but they are much less pronounced in the later stages. They are apt to simulate a rheumatic attack, with malaise, fever, muscular pains, anorexia, chills, and possibly sweating. In from a few days to a week spinal symptoms reveal themselves, the motor generally appearing before the sensory symptoms, though they may be contemporaneous, or the sensory symptoms may even appear first. In any event, they are apt at first to be irritative. The limbs will feel tired and heavy and drag in walking, and tremors or twitching occur, even cramps, and later paralysis, partial or complete, in the region involved. The lower limbs may alone be involved, or when the lesion is in the cervical region paralysis and atrophy of the upper with a spastic condition of the lower extremities may develop. The breathing is generally diaphragmatic in cases in which the intercostal muscles are involved. If the lesion is still higher up, death will quickly take place from failure of respiration. Such cases, however, are more apt to occur in the type known as *disseminated myelitis*, in which bulbar symptoms are prone to appear. The sensory symptoms at first are those of a tingling or burning character, or formication. Later, certain or all forms of sensation may be lost, and, roughly speaking, the upper level of anesthesia corresponds to the level of the cord involved. This "boundary region" is apt to be hyperesthetic, and in it the "girdle-feeling" is experienced. The reflexes may be lost at first, but soon return, and become exaggerated below the lesion. The condition of the trunk-reflexes may enable one to locate the position of the cord-lesion. There is not much wasting of muscles, as a rule, nor does the reaction of degeneration develop, unless the lesion is in the lumbar or cervical cord, when both will occur. Loss of control of the bowel and bladder may be among the earliest symptoms, though this is not the rule. While superficial ulceration may occur in any neglected case, the most marked trophic changes take place in those in which the lumbar cord is involved, either directly or by extension. In such cases, despite the most assiduous attention, extensive bed-sores develop. The course of the disease depends on the cause and the extent of the lesions. Death may occur in a few weeks from exhaustion, heart or respiratory failure, or from kidney-disease secondary to cystitis. Recovery is the rule, though with more or less permanent damage due to degeneration of some of the paths of conduction.

**Diagnosis.**—The distinction from hemorrhage into the cord or membranes has already been mentioned. From Landry's paralysis it can be separated by a reference to the subjoined table:

ACUTE MYELITIS.	LANDRY'S DISEASE.
Paralysis is sudden and generally becomes complete.	Paralysis begins in the feet and rapidly spreads to the muscles of respiration and deglutition.
Wasting and bed-sores are marked.	Trophic disturbances are absent.
Reactions of degeneration are distinct.	No reactions of degeneration.
Early involvement of the sphincters.	Bladder and rectum are not involved.
Girdle-pains sometimes mark the height of the lesion.	Girdle-pains are absent.

*Anterior poliomyelitis* is not accompanied by sensory symptoms. In *peripheral neuritis* pain of a shooting character is more apt to be present, and is almost invariably the first symptom to appear. Motor symptoms may not appear for some days. This is not the case in myelitis. In *compression of the cord* sufficient collateral evidence can usually be obtained to differentiate it from myelitis. *Hysterie paraplegia* is occasionally misleading. The character of the patient and the previous history should be thoroughly considered; moreover, in this form there are no trophic changes, and as a rule no bladder-symptoms; at any rate, there is no cystitis. Retention of urine may occur, but not incontinence. The diagnosis of myelitis can usually be made without great difficulty from the motor and sensory symptoms, the preservation of the knee-jerk, the vesical, rectal, and trophic symptoms, and often from the presence of the girdle-sensation in addition.

**Prognosis.**—The most acute cases are fatal in from three days to a week. Less acute cases generally recover with more or less loss of motor power.

**Treatment.**—Very little can be done to arrest the process in acute myelitis. The actual cautery should be tried as a counter-irritant, or an ice-bag may be applied to the spine. The patient should be placed on an air- or water-bed. Trophic changes should be looked for daily, and at the first sign of their appearance alcohol or some stimulating liniment should be employed. If the skin is broken, absolute cleanliness must be observed, and the wounds dressed antiseptically. It is well, also, to change the patient's position from time to time to avoid too long-continued pressure in any one spot. Ergot or ergotin should be given internally, and, especially in specific cases, potassium iodid. A general tonic and supportive treatment is indicated, and later massage, electricity, and baths.

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## CHRONIC MYELITIS.

THAT there are both a subacute and a chronic form of myelitis is generally conceded, though these types are not sharply circumscribed. As has been previously mentioned, it is quite likely that many cases exist in which the clinical symptoms do not seem to warrant the diagnosis of

myelitis, and yet extensive areas of degeneration may be found *post-mortem*. Even some cases of supposed hysteria may have a distinct pathology.

**Pathology.**—The lesions are most apt to be disseminated or diffuse, though there may be a single focus. Histologically, the chief differences from the acute variety consist in the greater amount of sclerosis, the thickened blood-vessels with contracted lumen, and an entire absence of recent hemorrhage. In some cases also the pia is much thickened in patches and firmly adherent. The nerve-cells are either seen to be in advanced stages of degeneration or they have actually disappeared. Secondary degenerations, above and below, proceed from the primary foci.

**Etiology.**—Any of the causes capable of giving rise to acute myelitis may cause the chronic variety, either by acting slowly over a long period of time or by their influence upon a person whose tissues are resistant. A process originally acute may become chronic, or a succession of acute attacks may give rise to a chronic condition. Gout, alcohol, and syphilis seem especially prone to cause chronic lesions. The condition may also be secondary to meningitis and to certain toxic blood-conditions other than those that have been mentioned.

**Symptoms.**—Any symptom occurring in the acute may be duplicated in the chronic form, though the onset of the latter is gradual. The symptoms are more or less obtrusive according to the region of the cord that is affected. If the cervical and lumbar regions are not implicated, no definite symptoms will be present, and probably there will be nothing more than subjective sensations and progressive weakness, with possibly some muscular wasting. The most characteristic features of a well-marked case are the irregular and successive involvement of various parts. There will be motor weakness, possibly of an arm, followed sooner or later by sensory impairment. Then one of the lower extremities may become involved, and ultimately paralysis will supervene. When the lesion is single this irregular onset is less apparent. In chronic transverse myelitis of the lumbar region, for instance, there will be paresis of the lower extremities, simultaneously or successively involved. The onset, however, is gradual, and months may elapse before the paraplegia will be complete. A girdle-sensation is apt to be present, together with lowered sensibility, and loss of sensation is very rarely absolute. The knee-jerk is increased, ankle-clonus is present, and in time the muscles become spastic. The sphincters are frequently implicated. Atrophy of the muscles is most pronounced when the anterior gray matter of the cervical or dorsal region is involved, but this may occur in any case. The reactions of degeneration can rarely be elicited.

**Diagnosis.**—The gradual, and in many cases the irregular, onset characterize this disease. In its various phases it may simulate almost any spinal-cord disease, and it is most apt to be confounded with tumor, pressure (carius or malignant), primary lateral sclerosis, progressive muscular atrophy, and syringomyelia. *Pressure*, whether due to a tumor, to caries, or to malignant disease, is apt to cause pain radiating in character, and the last two usually present collateral evidences in the deformity and cachexia. The symptoms, too, are always bilateral, while those of myelitis may be unilateral. From *progressive muscular atrophy*



it may generally be diagnosed by the irregular course it pursues. Apart from the painless ulcerations and the dysesthesia that usually occur in *syringomyelia*, it may be impossible to diagnose it from the latter disease.

The **prognosis** is necessarily grave. Recovery may be possible, but it is extremely rare. The process, however, may be arrested in some cases, and the strictly focal forms are less apt to prove fatal than the disseminated or diffuse.

**Treatment.**—More can be expected from general hygienic measures than from the use of drugs. In the early stages rest is indicated, but it is well also to employ passive exercise, to prevent, if possible, a too great contraction of the muscles. As soon as expedient—each case being judged on its merits—the patient should be taken out of doors. Change of air and of scene is advisable, as are also baths and massage. Mild counter-irritation may be applied to the spine, but care should be taken to avoid the areas of anesthesia. General tonics, iron, quinin, arsenic, and strychnin, should be given, also mercury or the iodids. The greatest possible care of the bladder should be taken in order to avoid cystitis.

## ANTERIOR POLIOMYELITIS.

### ESSENTIAL PARALYSIS OF CHILDREN.

(*Atrophic Spinal Paralysis.*)

**Definition.**—A febrile disease of more or less rapid onset, associated with muscular paralysis and atrophy, occurring chiefly in children, and most frequently in those under three years of age.

**Pathology.**—The condition is generally unilateral, and is a true focal myelitis; hence we find congestion, softening, and even cavity-formation. Microscopically, the chief feature observed is the destruction of the multipolar ganglion-cells of the anterior horn. If the examination is not made until months or years have elapsed since the onset, the condition will be about as follows: More or less asymmetry of the cord in the region affected, with sclerotic changes at the site of the lesion, and probably in the pyramidal tract also. The anterior nerve-roots of the same side will be found atrophied, and the muscles wasted, having undergone fatty degeneration and fibrous change.

**Etiology.**—The precise cause is not known, but the following predispose to the affection—viz. age, exposure, acute diseases (particularly those known to be infectious), and warm weather. The disease may occur at any age, but by far the greatest number of cases occur before the third year of life; they are about equally distributed between the two sexes. Later in life the condition is more common in males, chiefly between the ages of ten and twenty-five. It is rare after this period. Epidemics have been described, and, notably, one occurring during the summer of 1894. Dr. Caverly of Rutland, Vt., then reported 126 cases occurring in Otter Creek Valley, a limestone region of Vermont. At the

same time domestic animals—horses, dogs, and hens—were affected with a paralytic disease, this fact still further supporting the idea of an infectious origin. A similar epidemic has occurred in Ohio.

**Symptoms.**—The onset is generally acute, and may be sudden, in which case it is due to hemorrhage. Such cases do not strictly belong to this category, but they have been included, since the nervous symptoms are similar. Constitutional symptoms are absent as a rule. Moreover, when prodromal febricula precede an explosive onset of paralysis (hemorrhagic), we are justified in regarding it as a case of poliomyelitis. Generally, the sequence is as follows: Fever (usually slight), malaise, possibly vomiting (especially in children), muscular twitching, headache, and restlessness. In a few hours, or after one or two days, paralysis supervenes and quickly spreads, involving a greater or less area; it then remains stationary for from two or four days to from five to eight weeks, when improvement takes place, beginning in the part last affected. In some cases, after a most trifling indisposition over night, paresis is met with in the morning. In a few weeks only that portion remains paralyzed that is to be permanently damaged. Wasting of the muscles will be noticed a week or two after the onset of paralysis; these become flaccid and give the reactions of degeneration. Sensory symptoms are very rarely present—so seldom, indeed, that they need not be reckoned with. The reflexes are lost, both superficial and deep, and later contractures develop and result in various deformities. The growth of bone is seriously impaired in some cases. Complete recovery rarely takes place, nor is it to be expected when we consider the destruction of the neuron-body.

**Diagnosis.**—Usually this is not difficult, except, possibly, for the first few days in some cases. Close scrutiny will enable one to differentiate between this disease and a pseudo-palsy the result of pain on active or passive motion, as seen in rickets, scurvy, and in hip-joint disease.

**Prognosis.**—Some impairment of motion and more or less wasting of the muscles almost invariably remain. Danger to life, however, is very remote, though the subjects of infantile paralysis are predisposed to intercurrent affections, since their natural degree of resistance is lowered. The more rapid the loss of faradic irritability the less the extent of recovery.

**Treatment.**—I think we are justified in regarding this disease as infectious, probably caused by a specific micro-organism, although it is possibly an auto-intoxication. If this is granted, it behooves us to act promptly and render the emunctories in good condition. If the case is seen early, a few doses of calomel may be given, and these followed by a saline. Copious enemata of boiled water thrown high up into the bowel should also be employed. Should the fever be high, it must be met, as in any other case, by sponging or even by a cool bath, cold compresses to the head, and internally by the bromids, aconite, and the spirits of nitrous ether. During the febrile stage, or at least for a few days or a week, it is advisable to keep the patient in bed. The affected parts should be wrapped in cotton, and counter-irritation may be applied to the spine. As soon as possible the child is to be taken into the fresh air. It is of vital importance to keep up the general systemic tone, and hence the necessity for fresh air, change of scene, and for nourishing but easily digestible food. During this period massage and electricity should be employed, together with the administration of strychnin. In the later stages, when

contractures have set in, mechanical appliances may be necessary to correct deformity and to give support.

#### ACUTE, SUBACUTE, AND CHRONIC POLIOMYELITIS IN ADULTS.

1. **Acute atrophic spinal paralysis of adults**, as the acute form is called, has essentially the same symptomatology as the corresponding disease in children, except that the onset is apt to be more pronounced. Convulsions, however, scarcely ever occur. When pain is a prominent symptom we should be guarded in making a diagnosis. Initial pain is significant of a nerve-lesion, particularly if sensory disturbances can be found, and such cases would indicate a neuritis and not a poliomyelitis. Presumably the incidence of the poison has been on the axon, and not on the neuron-body, this view being consonant with the complete recovery that is sometimes seen in adults. When true poliomyelitis has existed complete recovery probably never occurs.

2. The **subacute form** has been described by Duchenne as "*paralysie générale spinale antérieure subaiguë*." It comes on, as a rule, without apparent cause, and the initial symptoms are very slight. In a few weeks failure of power is noticed in the limbs and paralysis gradually supervenes. After lasting for some time partial recovery follows, the paralysis and muscular atrophy remaining in a limited region only.

3. That **chronic poliomyelitis** exists has been proved by Oppenheim and other observers; yet it is probable that most cases described under this heading have been due to peripheral and not to central lesions. In neuritis, however, the paralysis is either unilateral or bilateral, and in the latter case it is symmetric, differing in this point from the irregular distribution of centric disease. Pain is common, and there is also tenderness along the nerve-trunks as a rule.

Recovery from neuritis may be perfect; at all events, it does not present the tendency that is met with in poliomyelitis to clear up perfectly, except in a limited area.

**Treatment.**—The general line of treatment that I have given for the infantile type is equally applicable in these forms. Ergot and belladonna may be used in the early stages, and, later, mercury or the iodids in small doses. Electricity and massage are of the greatest value.

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#### ABSCESS OF THE SPINAL CORD.

It is rare for inflammation of the cord to give rise to pus, yet a few cases have been described. The suppuration is necessarily micro-organismal in origin, and as a rule is either due to some septicemia or traumatism, or secondary to purulent meningitis. The *symptoms* are those of myelitis, but may be masked by any associated condition.



## UNILATERAL LESION OF THE SPINAL CORD.

*(Brown-Séquard's Spinal Paralysis.)*

THIS is not a distinct disease, but rather a grouping of certain symptoms, first studied by Brown-Séquard,<sup>1</sup> and hence bearing his name. It

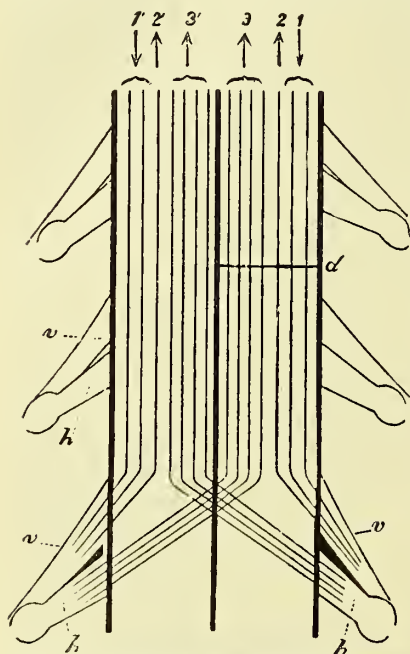


FIG. 74.—Schematic representation of course of main tracts in the cord, represented for a single pair of roots (Erb): *v*, anterior roots; *h*, posterior roots; 1, paths for motor and vaso-motor conduction; 2, paths for muscular sense; 3, paths for cutaneous sensibility on the right; 1', 2', 3', the same paths on the left. The arrows indicate the direction of physiologic conduction.

is met with particularly as a result of injuries (knife-thrusts and the like), though it may also be due to tumor or caries of the cord, to syphilis, or to any process causing compression of one-half of the cord. Such lesions intercept the motor impulses of the same side; the fibers having crossed in the medulla, the sensory fibers cross in the cord soon after entering, and hence sensation will be absent on the side opposite to the lesion (*vide* Fig. 74). A lesion in the cervical cord above the arm-nuclei causes motor paralysis of both arm and leg of the same side (spinal hemiplegia) and sensory paralysis on the opposite side. If in the dorsal or lumbar cord, the leg on the corresponding side is paralyzed, while that of the other is anesthetic. Lesions are seldom strictly confined to one side of the cord, but overlap a trifle, so that there is apt to be some loss of power on the anesthetic side; this, however, may be due to the recrossing of a few motor fibers at a lower level. The side of the

lesion is hyperesthetic—a fact for which no satisfactory explanation has ever been advanced. Muscular sense is diminished or lost on the same side. Above the hyperesthetic region an area of anesthesia commonly exists, and above this, again, an area of hyperesthesia. The reflexes are increased on the side of the lesion (inhibition being removed), and the temperature of that side is usually higher. On the anesthetic side the motor power, reflexes, muscle-sense, and temperature are all normal. Sometimes the sensory symptoms are limited to loss of pain and temperature sense.

<sup>1</sup> *Med.-Chir. Trans.*, 1889.

## LOCOMOTOR ATAXIA.

*(Tabes Dorsalis; Posterior Sclerosis.)*

**Definition.**—A systemic sclerosis affecting the posterior columns of the cord. In many cases foci of degeneration occur in the basal ganglia. The disease is characterized by a loss of coordination, absence of the knee-jerk, fulgurant pains, and the Argyll-Robertson pupil.

**Pathology.**—Macroscopically, it may be observed—1. That the posterior roots are more or less atrophied and grayish in color.

2. There is a thickening and adhesion of the spinal membranes, with some degree of congestion, particularly noticeable in the posterior region (not a constant change).

3. There is a slight change in the shape of the cord, and the affected regions assume a grayish tint. Change of color is well seen after the cord is hardened. Microscopically, degeneration of the peripheral sensory nerves will be found in certain cases to be more marked at the periphery and to diminish as the main trunks are reached. Rarely, changes in the motor nerves will be met with also, but only in cases in which the anterior horns are affected. The spinal ganglia are usually normal.

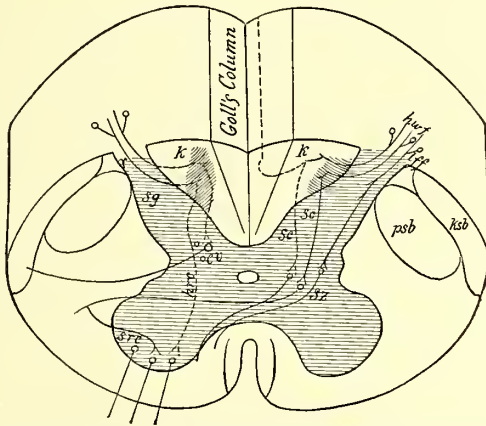


FIG. 75.—Diagram of primary degeneration-areas and secondary degeneration of the fibers in the beginning stage of tabes (Leube): *psb*, pyramidal tract; *ksb*, cerebellar tract; *hwf*, posterior root-fibers; *lff*, lateral entrance of delicate root-fibers; *k*, area of earliest degeneration; *r*, marginal zone; *sg*, substantia gelatinosa; *cv*, Clark's columns; *i*, anterior zones (remaining free); *sc*, sensory collateral fibers; *hrc*, collateral reflex of posterior column; *src*, collateral reflex of the lateral column; —, healthy fibers; ----, degenerated fibers.

4. There are degenerative changes in the posterior, and occasionally in the anterior roots (*vide* Fig. 75).

5. Cord-changes are present, consisting in the early stages of a degeneration of the fibers of the Spitzka-Lissauer column, of the post-root zone of Charcot, of the fibers going to the column of Clark, and of the comma tract. As the disease progresses more and more of the posterior columns—Goll and Burdach—is claimed, with the fibers of Gowers' column, the intermedio-lateral tract, and even the direct cerebellar tract.

This latter is only affected, however, when the cells of Clark's column are involved.

While the chief incidence of the poison, whatever this may be, is upon the nerve-fibers, yet we do meet with cases in which the posterior root-cells are diseased; as already stated, the cells of the anterior horn may be diseased also. There is an overgrowth of neuroglia that takes the place of the degenerated fibers, and when the membranes are thickened the strands of connective tissue dipping into the cord take on added growth.

6. There are cerebral and medullary changes. There may be some change in the nuclei of the columns of Goll and Burdach and in those of some of the cranial nerves. In addition to changes in the nervous system, certain cases present some morbid condition of the osseous system, consisting of erosion of the interarticular cartilages and atrophy and absorption of the bony articulating surfaces.

**Etiology.**—*Race.*—White races are more susceptible than negroes; and the disease is less frequently met with among the Jews than among other white classes. *Sex.*—Males are more liable to the disease than females, in the proportion of 10 to 1. *Age.*—Most common between the ages of thirty and forty. *Syphilis.*—Since Fournier in 1875 first pointed out the relationship between these two diseases, the opinion has steadily gained ground, despite the view of Leyden and other German authorities, that a large majority of tabetic cases (observers differ as to the proportion) have an antecedent history of syphilis. It must be clearly borne in mind that locomotor ataxia is not syphilis of the cord and brain, but a distinct entity, in most cases of which, however, syphilis stands as a predisposing factor. It will be remembered that in the description of anemia of the cord, lesions resembling those of tabes are found as a result of various toxemias, and it was suggested that this might throw some light on those cases in which no syphilitic history can be obtained. Exposure and sexual excess are possible factors; likewise traumatism. Alcohol is said to cause tabes, but this is very doubtful; it may certainly give rise to pseudo-tabes, the peripheral form. In England, Gowers has noted that locomotor ataxia occurs more frequently among urban than among rural populations.

**Symptoms.**—These may be grouped according as they occur in the early or late stages. The early or preataxic stage is one of variable duration; lasting, possibly, but for a few weeks in some cases; but as a rule it is distinctly chronic, even extending over many years. During this time pains of a peculiar type (fulgurant) develop. They are sharp and shooting, of sudden onset, and of just as sudden cessation; they do not recur in precisely the same place, but may occur in any part of the nervous supply of the affected region of the cord. Herpes may appear along the course of the nerves. The knee-jerk is either diminished or absent in by far the largest number of tabetics, though should hemiplegia occur later it will reappear.

*Ocular symptoms* are characteristic—the myosis and the absent light-reflex, with normal response to accommodation, constituting the Argyll-Robertson pupil. Other ocular symptoms may be present, however, and one of the earliest to develop may be strabismus with or without ptosis.



Diplopia may be the first evidence pointing to ocular involvement. Other eye-muscles may be affected also, producing ophthalmoplegia. Atrophy of the optic disk may be noticed at this stage. It usually begins as a circumferential change, and only gradually encroaches on the center; hence vision may not be noticeably impaired for some time. If an examination be made, however, during this period, it will be found that the field of vision is contracted. Rarely the auditory nerve becomes diseased, causing deafness.

After a variable period of time *certain motor symptoms* are super-added. The patient may notice that he experiences some difficulty when walking in the dark. He will stagger or stumble, or, while washing his face, he may observe that he cannot balance himself properly with his eyes closed. Romberg's sign can now be elicited—viz. when the eyes are closed, and particularly if the feet are held close together, it will be noticed that station is imperfect. Later he finds that even in the daylight he has difficulty in maintaining his equilibrium. At first he cannot stand with his feet close together. This difficulty is greatly accentuated when the eyes are closed. Ere long the *characteristic gait* is manifest. The legs are spread wide apart, the patient leans forward, using one or even two canes, and with eyes fixed upon the ground a few feet in front of him throws one leg around, at the same time lifting the foot higher than is really necessary and bringing it down on the heel. As a general rule there is no muscular wasting, and hence there is no loss of motor power. A certain degree of incoördination of the arms is present in many cases, but is unobtrusive.

*Sensory Symptoms.*—Apart from the pains already noted, these consist of paresthesia, numbness, tingling, burning; anesthesia and hyperesthesia of irregular distribution; retardation of the transmission of sensory impulses; in some cases a peculiar condition in which a pin-prick on one leg, for instance, will be referred to the other (*allochiria*), or in which one point of contact made by some one is felt in many places at once (*polyesthesia*). Usually the patient feels as though he were walking on cotton or felt. Muscle-sense is more or less impaired in every case; hence the difficulty experienced by these cases in recognizing any position in which a limb may be placed. Sexual power is usually lost early.

*Certain visceral symptoms, or crises*, as the French term them, are prone to occur. They are chiefly gastric (sometimes accompanied by vomiting of acid material), but laryngeal, nephralgic, and rectal crises have also been described. The pain is usually intense. Constipation is the rule, though in some cases incontinence of feces occurs, particularly if the stool is loose. There may also be retention of urine, with incontinence.

*Trophic Changes.*—Apart from the herpes previously mentioned, the most striking trophic changes are those occurring in and around the large bony joints (the so-called tabetic arthropathies). Special attention was called to these by Charcot; they are not a common condition, and are probably due to the influence upon the nerves that supply the joints. Occasionally the condition would appear to be excited by traumatism. The affected joints are not painful; they may be the seat of exudation which is rarely purulent. *Arthropathies* may supervene at any period

of the disease, even the preataxic. These conditions affect primarily and chiefly the bones and cartilages entering into the larger joints. The involved osseous tissue becomes atrophied, brittle, and is finally destroyed. Muscular wasting is rare as an early condition, though it may occur later; it is due either to neuritis or to involvement of the anterior horns.

Since the disease does not of itself prove fatal, these symptoms may last for years and the patient eventually die of some intercurrent affection. In other cases paralysis finally develops and the patient becomes bedridden. Hemiplegia may develop as a complication at an advanced stage, as may general paralysis or other forms of nervous disease.

**Course.**—Rarely the disease runs a very rapid course. The preataxic symptoms—pain, loss of knee-jerk, Argyll-Robertson pupil with or without ptosis and diplopia—may only exist a few weeks before incoördination develops. The latter will then reach its acme in twenty to thirty days. This is very unusual, however. As a rule, the first or preataxic stage extends over a period varying from months to even as long as twenty-five years. Dr. Wm. Egbert Robertson has related to me the case of a man aged fifty-eight who for fifteen years has had fulgurant pains and an absence of the knee-jerk, but neither ocular nor any other symptoms. In some cases the first stage may be absent. The second or ataxic stage—that of incoördination—is generally slowly progressive, finally reaching a point at which it remains; rarely, more or less improvement may follow. When optic atrophy develops, ataxia either does not appear, or, having done so, fails to advance. The final stage in a few cases is only reached when the patient has become paralyzed and bedridden.

**Diagnosis.**—This is readily made when we have a combination of the absent knee-jerk, fulgurant pains, and the Argyll-Robertson pupil. However, the loss of knee-jerk, associated with one of the other symptoms in an otherwise healthy man, is, to say the least, highly suggestive of the disease; the addition of incoördination serves, of course, to clinch the argument.

**Differential Diagnosis.**—*Peripheral Neuritis.*—The symmetric distribution of symptoms, tenderness in the muscles, frequent herpetic rashes, motor weakness and wasting, pain (not fulgurant in type), greater prominence of parasthesia, absence of the Argyll-Robertson pupil, knee-jerk often increased (absent in diphtherial form, but other symptoms and history serve to distinguish it), and later, either diminished or absent, and the history of the case, are sufficient. *Alcoholic* and more rarely *arsenical* poisoning give rise to a condition closely resembling true tabes in that there is the loss of knee-jerk, often sharp pain, and incoördination, though the latter symptom is never as marked as in advanced tabes. The gait, however, is totally different, and consists of the high “steppage” gait described in the discussion of *Peripheral Neuritis*.

*General paralysis of the insane* may present much difficulty. Spinal symptoms may occur in general paresis, and conversely in certain cases of tabes symptoms of general paresis develop. Time alone will solve the problem.

*Ataxic Paraplegia.*—Apart from the absence of pain and anesthesia, incoördination is followed by a spastic condition. The knee-jerk is much exaggerated and the so-called ankle-clonus develops.

*Cerebellar Disease.*—The incoördination does not resemble that of ataxia; optic neuritis is present; also headache and vomiting appear in well-marked cases. The knee-jerk is always present.

There are certain conditions, already described under *Anemia of the Cord*, in which lesions of the posterior columns of the cord occur. Some of them are very much like tabes, but do not present the "combination of symptoms" seen in locomotor ataxia. As a rule, the Argyll-Robertson pupil is absent, and less frequently the lightning pains also.

The crises may be mistaken for *disease of the various organs involved*. Repeated attacks of acute pain, tabetic in character, and particularly in adult males, should, however, excite suspicion, and an absence of the knee-jerk and other characteristic evidences will always be present in ataxia.

When the chief lesion is in the dorsal region the pain may be mistaken for that of *spinal caries* or even *neuralgia* or *rheumatism*. From caries it may be differentiated by the fact that in vertebral disease the pain is more or less localized, and that it is much increased by movements. Moreover, the other symptoms of ataxia are wanting—*e. g.* ocular troubles, incoördination, and absence of the knee-jerk. The latter point also holds good in cases of rheumatism and intercostal neuralgia. For the diagnosis from hereditary ataxia *vide* p. 1084.

**Prognosis.**—The outlook is not particularly bright. While, as already stated, the disease does not cause death, perfect recovery is never obtained. Of course the prospect is much brighter the earlier the case is taken in hand, and some improvement may be expected in most cases. The fact that the patient has had syphilis does not modify the prognosis one way or the other.

**Treatment.**—Rest (first suggested by Weir Mitchell) is imperative when the patient commences treatment, and especially when pain is early complained of, massage and electricity being employed meanwhile to keep up the tone of the muscles. In my opinion the rest-treatment retards the progress of ataxia more effectively than any other measure, but it cannot be used with the expectation of producing a cure. The bowels should be moved daily, and the urinary functions especially looked to. In certain cases catheterization is necessary. The patient should then be taught, first, what surgical cleanliness means; and secondly, how to use the instrument. Counter-irritation along the spine is of very little more value than suspension. The diet should not be heavy, and if gastric crises occur special care should be taken in this direction.

In cases giving a previous history of syphilis, mercury and the iodids should be used; it is, however, doubtful if they are of any direct benefit, although when the venereal disease is of comparatively recent date some improvement in the tabetic lesions is possible. Potassium iodid should be used freely. Mercury is best introduced into the system by inunction, and it is my custom to order one dram to be rubbed into the arm-pits, flanks, or inner surfaces of the thighs daily until the gums show the specific influence of the remedy. Then the inunctions are discontinued, and the potassium iodid and mercuric chlorid are administered in combination three times *per diem*. The dose of the latter remedy should be small ( $\text{gr. } \frac{1}{24}$ —0.0027), but the iodid may be used in ascending dosage.

Electricity is of doubtful utility. The galvanic current is to be



chosen, and Erb advises placing the medium-sized cathode over the cervical sympathetic, and the larger anode near to the spinal column on the opposite side, moving it at brief intervals in the downward direction. This method must be continued for many months. Frenkel has recently advocated the systematic education of the muscles in coördinated movements. Hydrotherapy is a serviceable measure if judiciously employed. Neither cold nor hot baths are free from deleterious effects, but tepid baths ( $80^{\circ}$ – $90^{\circ}$  F.— $26.6^{\circ}$ – $32.2^{\circ}$  C.), combined with gentle friction of the body-surface, are signally useful. Among the numerous natural springs enjoying more or less popularity there are two in especially high favor—the carbonic-acid thermal saline springs of Oeynhausen-Rehme in Minden and Aix-la-Chapelle in Germany. The chief benefit may, after all, be credited to the invigorating effect of the changed environments.

The fulgurant pains, or those of the various crises, are occasionally so severe as to require bromids, codein, or even morphin, though the use of the latter agent is always to be postponed until other means are exhausted. Antipyrin or salol and phenacetin may also be tried in this connection. In any case the patient should live a simple, regular life, avoiding excesses of all kinds, and particularly sexual and alcoholic indulgences.

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## HEREDITARY ATAXIA.

(*Friedreich's Disease.*)

**Definition.**—An hereditary disease, first described in 1861 by Friedreich. The symptoms are primarily manifested in early life, and the disease is characterized by ataxia, defective speech, nystagmus, absence of the knee-jerk, and more or less secondary deformity, as spinal curvature or talipes.

**Pathology.**—The postmortem findings are essentially those of locomotor ataxia and ataxic paraplegia. The spinal membranes are somewhat thickened and adherent, especially over the posterior part of the cord, and that, too, chiefly in the lumbar region. The posterior nerve-roots are generally atrophied and sclerosed. The columns of Goll and Burdach are degenerated, particularly in the lumbar region, and to a lesser extent in the cervical. Degeneration is also found in the lateral, and to a slight degree in the anterior, columns. The chief microscopic change is a marked neuroglial overgrowth, as shown by Déjerine. The nerve-cells of the cord are generally normal. Frequently the cord is abnormally small. Cerebral lesions also have been found in this disease.

**Etiology.**—1. Family tendency (heredity) has a strong influence. A single case, however, may develop in a family.

**Age.**—Most commonly the disease appears between the third and twelfth years, though it may appear earlier.

Infectious fevers (in particular) and other acute diseases frequently precede the evolution of this complaint. Trauma and many other conditions have been described as exciting causes.

**Symptoms.**—The first evidence of the disease is impaired coördination, first in the legs, and, later, in the arms; it is most marked when the eyes are closed. Attention is often called to this symptom by the fact that the child stumbles, ambles, and staggers, and cannot walk properly. The gait, however, lacks the pronounced stamp of true ataxia. Rutimeyer has pointed out that in many cases the great toes are turned upward. Some children never learn to walk. Romberg's symptom is generally present. Movements of the arms, when these are ataxic, are irregular and jerky, and jerky movements of the head may also be observed. Bilateral nystagmus develops and the speech becomes affected. At first there is a mere impediment (a stuttering), but later syllables, or even whole words, are omitted and an unintelligible jargon results. The knee-jerks are almost always absent. There is no optic atrophy, nor are any sensory symptoms present as a rule. The sphincters are normal. There are no trophic changes in the skin or the joints, and no visceral crises. Vaso-motor symptoms—flushing, sweating—are sometimes observed. There is no mental change.

Talipes and spinal curvature are generally met with after the disease has existed for some time. In old cases muscular weakness and wasting are present, but there is no electric change in the muscles.

The **course** is always slow. It may last for many years, thirty or even more.

**Diagnosis.**—Usually this is not difficult, and especially when more than one case exists in a family. The age, incoördination, shambling gait, nystagmus, scanning speech, and deformity are strikingly characteristic.

**Differential Diagnosis.**—*Locomotor ataxia* appears later in life, and the preataxic stage (pain, absent knee-jerk, and ocular symptoms) is generally well marked. It is absent in hereditary ataxia, nor does the latter present the sensory and visceral symptoms met with in the true form. Further, the gait is very different.

*Ataxic paraplegia* shows an exaggerated knee-jerk, the presence of ankle-clonus, and an absence of the ocular symptoms, nystagmus, and the scanning speech.

*Disseminated Sclerosis.*—Tremors are almost always present, but these are fine and never coarse as in hereditary ataxia. There may be nystagmus, incoördination, and imperfect articulation, but the cases are isolated (*i. e.* they do not run in families).

The **prognosis** is necessarily bad. The disease is progressive, though it does not kill directly. It may last thirty years or more.

**Treatment.**—Little or nothing can be expected from it. The same general treatment should be pursued as for locomotor ataxia.

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## SPASTIC PARAPLEGIA.

(*Primary Lateral Sclerosis; Spastic Spinal Paralysis.*)

**Definition.**—A disease of the spinal cord characterized by loss of power, contractures, exaggerated reflexes, a peculiar gait, and by precipitate micturition. Spastic paraplegia (spasm plus motor paralysis) is

met with as the result of the various pathologic substrata. Any transverse cord-lesion above the lumbar region may cause motor paralysis, spasticity, exaggerated knee-jerk, and ankle-clonus. The same condition results from a lesion in any part of the upper segment, from the cortical motor cells to the terminal arborization of the axon in the cord.

It is believed that fibers of the pyramidal tracts may be primarily involved, and, since they course chiefly through the lateral cord-region, the resulting condition has been named primary lateral sclerosis. This is purely hypothetical, however, for only two uncombined cases have been found. This may be due to the fact that the disease does not tend to shorten life, and that therefore the same condition that caused degeneration of the pyramidal fibers may subsequently act on other fiber-systems. Since in the case of the lower segment it is the peripheral portion of the axon that, in many cases at least, first yields to the morbid influence, so may it be with the upper segment. In such an event, however, the degeneration would be an ascending one, and the converse of that which is usually met with in the motor tracts. Thus we see that the same clinical condition may be etiologically quite different. The following are the chief varieties :

#### PRIMARY LATERAL SCLEROSIS.

That this condition exists alone is questioned, as I have already stated. Von Stofella has reported a case, but no microscopic examination was made. Morgan's and Dreschfeld's case, published in 1881, seems to be the only one that may be regarded as a true type. The only pathologic change observed was in the pyramidal tracts of the anterior and lateral regions.

**Etiology.**—It is most apt to occur when there is a *neuropathic family tendency*. Age, generally between twenty-five and forty, exerts an etiologic influence. *Exposure, acute disease, and traumatism* are all predisposing causes. *Syphilis* has been said to predispose to the condition, but if so it is rather rare.

**Symptoms.**—In typical cases the onset is slow. The patient complains of feeling tired, and is less capable of exertion than formerly. Weakness of the legs develops, and with it increasing difficulty in walking. Even at an early stage some rigidity of the muscles will be present when the limb is extended; later this becomes a prominent symptom. The spasm is at first of little moment. It may only be noticed in the morning. When the disease has advanced, however, it becomes pronounced, so that it may not be possible to flex the limb, or, if flexed and an effort is made to extend it, it will often spring forward like a knife-blade in clasp-like rapidity. This spasticity is often so marked that in walking, so long as the ball of the foot touches the ground, clonic contractions occur; these also appear when the individual is in a sitting posture unless his legs are extended. The gait is characteristic; the legs are stiff, and move with an evident effort, while the toes scrape the ground. In some cases the adductor spasm is so great that the legs cannot only not be separated, but are actually overlapped in walking (*cross-leg progression*). In course of time the power of walking may be lost. The flexor muscles are usually weakened. The knee-jerk is very much



exaggerated, a mere tap causing a sharp, quick response. Ankle-clonus can always be elicited. Pain and other sensory manifestations are often absent, though dull and fleeting pains in the back and limbs may be complained of. The arms are frequently unaffected. The sphincters are rarely involved, and ocular symptoms do not occur, though nystagmus is occasionally met with. Seguin states that the ability to retain the urine is lessened and precipitate micturition results.

The **diagnosis** is not difficult. Certain hysteric cases may occasionally simulate it very closely, but these do not present the characteristic spasticity of the true form, nor is the knee-jerk increased quite as much, and ankle-clonus is either slight or absent. Then, too, in hysteria spots of anesthesia are commonly met with. Drs. Bastian and Russell Reynolds have described "paraplegia dependent on idea," in which no hysteric element entered.

#### SECONDARY SPASTIC PARALYSIS.

As I have already mentioned, transverse lesions above the lumbar region (caries, tumor, sclerosis, myelitis) are followed by degeneration of the pyramidal tracts, and as a result there are weakness in the limbs, increased reflexes, and more or less rigidity. In certain cases the latter may be absent, as Bastian has shown, and the limbs will be flaccid.

#### CONGENITAL SPASTIC PARAPLEGIA.

This condition, the symptomatology of which is practically that of the adult types previously described, is almost always the result of some injury at birth, either instrumental or due to a malposition, as first pointed out by Dr. Little and since abundantly confirmed by Spencer, Dr. Sarah McNutt, Sachs, and others. The disease is probably always due to meningeal hemorrhage. In recent cases more or less extravasated blood is always found over the central convolutions and often at the base. Later, cases show atrophy and sclerosis of the motor region, the blood having been absorbed. Nothing abnormal may be noticed for a few days or weeks, though rarely convulsions, or even bulbar symptoms, may early manifest themselves. Generally, the child is several months old when the mother first notices some impairment of movement, and not until the child tries to walk will she observe anything out of the way. The abnormality varies from a slight difficulty in walking, in which the toes barely scrape the ground, to a total inability to walk, owing to the high degree of adduction spasm. Between these extremes are various grades of talipes equinus and cross-legged progression. Sensation is usually normal. The bladder and rectum are not implicated. Some cases present evidences of impaired cerebral development—idiocy and imbecility. Some observers have also described what they believe to be an hereditary form of spastic paraplegia (notably Drs. Gee and Sachs).

#### ATAXIC PARAPLEGIA.

This name was given by Gowers to a condition in which spastic paraplegia and ataxia coexist, owing to simultaneous involvement of the lat-

eral and posterior columns. The posterior root-zones escape, and hence the retained reflexes. This same morbid condition may be met with in Friedreich's disease (hereditary ataxic paraplegia), or primary lateral or posterior cases may extend and involve the posterior or lateral columns respectively. Disseminated sclerosis may possibly present the same symptoms. The type Gowers describes occurs chiefly in males of middle age. Traumatism and exposure seem to predispose to the disease, as does syphilis very rarely.

*Symptoms.*—These develop insidiously. The patient tires rapidly, and some impairment of the power of walking is observed. In turning quickly he stumbles, and there is difficulty in walking in the dark, or even in standing when the feet are close together. The reflexes are increased at an early date, and spasticity supervenes and is progressive, though it never becomes as marked as in uncombined lateral sclerosis. The gait is somewhat similar to that met with in locomotor ataxia, but it lacks the forcible stamp already described. When the arms are involved the same ataxia, with weakness, spasticity, and increased reflexes, is met with. Sensory symptoms are generally absent, and fulgurant pains are never present. When pain occurs at all, it is of a dull character and often in the sacral region. Optic atrophy does not occur. Nystagmus is often seen, though other eye-symptoms very rarely appear. Sexual power is lost. The sphincters are not usually involved, though retention of urine may occur. Ultimately, the case generally partakes more of the nature of a lateral sclerosis, but the features of a posterior sclerosis may rarely predominate. Mental symptoms often develop in the late stages.

The *diagnosis* is easy in typical cases. The ataxia, with myotatic irritability and spasticity in the absence of sensory and ocular symptoms, is characteristic.

#### COMBINED SYSTEM SCLEROSIS.

Ormerod and Dana have published valuable treatises on this subject. In 1891, Dr. James Putnam of Boston described a group of system sclerosis, with diffuse collateral degeneration, occurring in enfeebled persons past middle life, and more particularly in women. He had had 8 cases, and made autopsies on 4. In the white columns of the cord he found both recent and old degenerations and disintegration of the cells of the gray matter. In 1 case he found some degeneration in the peripheral nerves. The chief symptoms were motor weakness of all four extremities, but especially the lower, with some impairment of sensation and general muscular wasting. In 3 cases there was an exaggerated knee-jerk with ankle-clonus; in 1 lancinating pains, and in another incoördination. The fatal cases ran a course of three or four years. Several of them showed lead in their urine, and Putnam thinks that this may have been an etiologic factor in some instances.

The *symptoms* of combined sclerosis partake of the nature of locomotor ataxia and spastic paraplegia, but are less marked than either of these diseases. The onset is slow, there is more or less incoördination, and Romberg's symptom can be elicited as a rule. There is loss of motor power, and the sensory symptoms are slight. There may be dull sacral pain. Optic-nerve atrophy very rarely occurs, though there are

certain eye-symptoms. The reflexes are generally exaggerated, and "ankle-clonus" is present.

The *diagnosis* is based upon the presence of paraplegia with increased reflexes, associated with sensory symptoms—paresthesiæ—and rarely pain.

#### REFLEX PARAPLEGIA.

Since this was at one time so warmly put forward by Brown-Séquard as a distinct entity, it seems justifiable to speak of it, though in the light of our present knowledge we are not disposed to give it any nosologic distinction. It was supposed to be due to anemia of the cord, and to be the result of irritation reflected from a sensory nerve to vaso-motor nerves. The so-called "urinary paraplegia" was included in this category.

#### INTERMITTENT PARAPLEGIA.

Romberg was the first to call attention to this condition. His original case was that of a woman aged sixty-four, in whom paraplegia developed suddenly with involvement of the sphincters. The sensations were normal. In about twenty-four hours she was so much better as to be able to walk; micturition was normal, but there was some weakness. Next day, however, the paraplegia returned. These attacks, with almost normal intervals assuming a periodic character, induced him to give quinine, which he did. Recovery was the prompt result. Erb and others have since reported cases, but it is now believed that they are due to involvement of the peripheral nerves rather than of the cord.

**Treatment of Spastic Paraplegia.**—In general the treatment is the same as that of locomotor ataxia. This is especially true if syphilis is suspected. Little can be done, as a rule, for the disease is usually progressive in spite of all treatment. Belladonna or hyoscin seems to lessen the spasm in some cases. Attention should be given to the bladder and bowel, particularly to the former. In the congenital form operative measures are often requisite to overcome deformity.

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## MULTIPLE SCLEROSIS.

(*Insular or Disseminated Sclerosis.*)

**Definition.**—A disease due to the development of sclerotic patches, occurring in an irregular manner throughout either or both the brain and spinal cord. It is characterized by paresis, intention-tremors, scanning speech, and mental disturbances.

**Pathology.**—The sclerotic tissue occurs especially in the white matter, though any part of the cerebro-spinal axis may suffer. The cortex is rarely implicated. The spots are usually well circumscribed, gray or grayish-red in color, and on section may be level with, raised from, or depressed beneath the normal line of section according as to whether it is in the early, hypertrophic, or cirrhotic stage. The cranial nerves may be involved at their origin, the first, second, and tenth being



particularly vulnerable. The medullary sheath of nerve-fibers in the affected region degenerates early, but the axons are markedly resistant. Since they are not cut off from their trophic center, secondary degeneration is rarely met with. The blood-vessels show more or less proliferation of the adventitia, and endarteritis is not an uncommon condition. Whether this vascular change is primary or secondary is unknown. Microscopically, the sclerotic areas are made up of an overgrowth of neuroglia-cells and fibers and of the ordinary connective tissue. In certain cases these patches exhibit some tendency to involve special parts of the nervous system, as the lateral or posterior columns.

**Etiology.**—There is no definite and known etiologic factor. Among the possible predisposing causes may be mentioned emotions, trauma, heredity, exposure, infectious and exhausting diseases of any kind, and perhaps hysteria. It is important to remember, moreover, that it is frequently impossible to diagnose this disease in its early stage from hysteria. This point is dwelt upon particularly by Buzzard and Bastian, and many cases of supposed hysteria have subsequently proved to be cases of multiple sclerosis. The difficulty is manifestly greater when the patient is a woman. Age and sex are also, in a sense, predisposing causes. The majority of cases occur between twenty and thirty years of age, though the condition may occur in children. Pritchard has collected over fifty published cases occurring between the ages of fourteen months and fourteen years, and about equally divided as to sex. Among adults disseminated sclerosis is met with somewhat more frequently in women.

**Symptoms.**—These may be described under two headings: first, the general symptoms, or those common to all cases of the disease, and not explicable from the position of the sclerosis; and, secondly, those dependent on the locality of the lesions. The disease is always chronic, and either remissions, or one or more intermissions occur, and in some cases may extend over several years. The first evidence of the disease is *loss of power*, first in one, then in the other, lower extremity. Later, paresis develops in the upper extremity. Sooner or later other general symptoms appear—viz. tremors, nystagmus, scanning speech, increased reflexes, and optic-nerve atrophy. The *tremor* is volitional (*intention-tremor*), and when the patient is at rest no abnormal movement is manifest, as a rule. On attempting to use the hands, or in walking, a fine, trembling motion of the limbs results. The head may be similarly involved, and some incoördination is commonly associated therewith. The *nystagmus*, too, is brought out when the eyes are in use. It is more marked in lateral than in vertical movements. *Speech* is slow and deliberate (staccato or scanning), the tendon-reflexes are increased, ankle-clonus may be present, and optic-nerve atrophy is of frequent occurrence. No alteration of sensation occurs, other than perhaps some numbness or tingling. There is no wasting of, nor electric change in, the muscles, nor do bed-sores occur. Vertigo is usually present. The mental phenomena are at first hysteroid, and they may never progress beyond this point. In other cases dementia, or even acute maniacal outbursts, are met with, but these are rare. During this stage epileptiform or apoplectiform attacks may occur. The symptoms directly resulting from the local lesions cannot be given in detail. Certain types result,

however, that depend upon the tendency of the sclerotic areas to involve certain tracts, and these are—first, a form resembling lateral sclerosis, due to implication of the lateral tract; and, secondly, a form similar to locomotor ataxia, in which the posterior columns especially suffer.

The **diagnosis** is generally easy after the disease has lasted some time. The intention-tremor and the gradual and progressive loss of power, with increased reflexes, scanning speech, and mental deterioration, are sufficient. The following table gives the differential points between this disease and *paralysis agitans*, *locomotor ataxia*, and *hereditary ataxia*:

DISSEMINATED SCLEROSIS.	PARALYSIS AGI- TANS.	LOCOMOTOR ATAXIA.	HEREDITARY ATAXIA.
Rarely occurs in children. Generally between the twentieth and thirtieth years.	Occurs in persons over forty years of age.	Rarely before the twentieth year.	Usually before the twentieth year. Generally affects several in the same family.
No sensory symptoms, as a rule. Sight may be impaired, the hearing less frequently. The Argyll-Robertson pupil is absent.	No sensory or special-sense symptoms of any importance. Argyll-Robertson pupil is absent.	Fulgorant pains an early symptom. Sight and hearing are commonly affected. Often diplopia and Argyll-Robertson pupil are present.	Sensory symptoms are rarely present. Diplopia and Argyll-Robertson pupil are absent.
Nystagmus is present, as a rule.	No nystagmus.	No nystagmus.	Nystagmus is frequent.
Reflexes are exaggerated; ankle-clonus is present. There may be muscular rigidity.	Reflexes are normal; very rarely they may be plus. Permanent muscular rigidity.	The knee-jerk, ankle-clonus, and rigidity are all absent.	The knee-jerk is lost in the course of the disease; it is rarely increased. No rigidity.
Scanning speech.	Speech is slow and deliberate on commencing a sentence, but soon it becomes hurried.	No speech-defects.	Speech is slow and irregularly scanning.
A tremor is generally present on voluntary movements only. If the tremor occurs during rest, it is fine. Oscillations of the head are frequent; of the trunk, less so.	Tremor when at rest. Voluntary movement may make it cease temporarily. The head may shake, with rather a vertical than an oscillatory movement.	No tremor. Incoördination is marked. No oscillations of the head or trunk. Romberg's symptom is present. Trophic disturbances are common.	Incoördination is present, but is not increased by closing the eyes. Static ataxia may be noted.
Mental disturbance is frequent.	No mental phenomena.	Mental disturbance is rare.	No mental disturbance.
Gait is usually spastic and parietic, and often uncertain.	The head is bent back and arched; the face is immobile and mask-like. The gait is propulsion, festination, retropulsion, or lateropulsion.	The gait is stamping in character; the legs are moved stiffly. There is difficulty in urination.	The gait is swaying and irregular, like that of a drunken man. The legs are not kept wide apart as in locomotor ataxia.

The **course** usually extends over five to ten or even fifteen years, and death is generally the result of some intercurrent affection, though it may occur during an apoplectiform or convulsive attack. Rarely it is due to failure of the heart or respiration.

The **prognosis** is always bad.

**Treatment.**—No remedy is of any avail. Silver nitrate, mercury, the iodids, and arsenic may be tried. Rest and easily assimilable food are also of prime importance.

#### PSEUDOSCLEROSIS.

In 1883 Westphal described a case characterized by disturbance of speech, slowness of the movements, decrease of both intelligence and irritability, apoplectiform attacks, pronounced tremor, spasticity and increased reflexes, slight disturbance of sensation, and no involvement of the sphincters. The autopsy was entirely negative. Since then similar cases have been reported, especially by Strümpell. The disease usually begins in childhood and causes death in the course of several years.

The *diagnosis* cannot be made from multiple sclerosis during life.

*Treatment* is useless.

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### BULBAR PARALYSIS.

(*Glosso-labio-laryngeal Paralysis.*)

**Definition.**—An acute or chronic disease, due to involvement of the motor nuclei of the medulla oblongata. It is generally secondary to some condition affecting other portions of the motor path, and is characterized chiefly by a difficulty of speech or of deglutition. Three varieties have been described:

1. **Sudden or apoplectiform**, this being due to hemorrhage, embolism, or softening. The onset is always sudden, often with vertigo, and possibly vomiting, with or without loss of consciousness. The power to articulate is impaired or lost. The lips and tongue are involved, and hence the pendulous lower lip, the dribbling of saliva, and the atrophy of the lingual muscles. There are dysphagia and generally frequent attacks of choking.

The *symptoms* are less characteristic than those of the degenerative form. They are less regular in type, and usually are widespread at first; later, some improvement takes place. In other cases, after more or less of a respite, degeneration sets in and they grow progressively worse.

The *diagnosis* of this type is not usually difficult. "Pseudo-bulbar paralysis" must be borne in mind, however, and is a condition due to a bilateral lesion of the motor cerebral cortex in the lower frontal parietal region or of the motor fibers in the course. There is great danger to life for some little while in these sudden cases. Later the prognosis is rather more favorable than in the other forms.

2. **Acute Inflammatory.**—Here the onset is less abrupt, requiring a



few days to a week to develop. But for this fact the symptoms are much the same as in the preceding form.

3. **Chronic Bulbar Paralysis.**—This condition occurs chiefly in males beyond middle life. The cause can seldom be discovered, though certain cases seem to be of toxic origin. It may develop in the course of progressive muscular atrophy, amyotrophic lateral sclerosis, insular sclerosis, or other disease of the cord.

The *symptoms* are bilateral, the tongue being usually the first to suffer. The patient may notice that he cannot speak for any length of time without fatigue, and that he will then articulate indistinctly. Soon he observes that there is a marked and progressive *impairment of speech*. The muscles of the lips and other muscles of the lower part of the face atrophy. He can no longer whistle. Speech is rendered still more defective, owing to paralysis of the lips. The lower lip drops, and the saliva constantly dribbles from the mouth and may be greatly increased in amount. *Difficulty in swallowing* is always present to a greater or less degree. Owing to the lingual paralysis, the tongue can neither be protruded nor can it be used to manipulate the food and make a bolus. It is atrophied and the mucous membrane is wrinkled. *Fibrillar tremors* are present. The *larynx* is involved, so that phonation is imperfect, but it is not so marked as the implication of other parts. Particles that enter the larynx cannot be ejected, owing to motor paralysis. There are no sensory symptoms, and the power of taste is normal. The mind generally remains clear, though the patient is often emotional, and cries or laughs without apparent cause. This type of bulbar palsy is particularly liable to develop in the course of progressive muscular atrophy. The *course* of the disease is slow, and death is usually due either to inspiration-pneumonia or to interference with respiration or circulation.

The **diagnosis** is not difficult, as a rule, the bilateral character of the symptoms rendering them distinctive. In the *pseudo-bulbar form* previously mentioned the limbs are often paralyzed also (double hemiplegia). *Tumors* rarely, if ever, give rise to such regular bilateral symptoms. I have met with 2 cases of chronic bulbar palsy, and 1 occurring in the course of Bright's disease, in which no postmortem lesion could be found that would account for the condition. In neither of the cases was there much atrophy, though otherwise they conformed to the regular type.

**Treatment.**—The disease is incurable. Hypodermies of strychnin, or of strychnin, morphin, and atropin, are of value in controlling the salivary flow. Electricity is of no value. Semi-solid food is probably the most readily taken, and it is often necessary either to use an esophageal tube or to employ rectal alimentation.

## AMYOTROPHIC LATERAL SCLEROSIS.

(Charcot's Disease.)

**Definition.**—A disease of the entire motor system, from the cerebral cortex to the muscles: characterized by loss of power, spastic symp-

toms, and muscular atrophy. The first clear and thorough description of the clinical symptoms and pathological anatomy was given by Charcot in 1872.

**Pathology.**—The pyramidal tracts are degenerated, the process commencing either in the cortex, crura, or medulla, and extending to the termination of the neurons in the cord. The ganglion-cells of the anterior cornua are atrophic, there is degeneration of the anterior roots and of the muscle-fibers, the blood-vessels in the affected parts are dilated, and in the early stages granular cells are present.

**Etiology.**—The disease is more frequent in males and usually begins in early adult life. Exposure has sometimes been noted in the previous history, but neuropathic heredity does not appear to have any influence.

**Symptoms.**—Three stages are generally recognized: (1) The involvement of the upper extremities. (2) The participation of the lower extremities. (3) The appearance of bulbar symptoms. At first there are weakness of the upper arms, atrophy of the muscles, and moderate exaggeration of the reflexes; in the course of a few months the symptoms of spastic paraplegia develop, all the reflexes are greatly increased, and there are chin- and ankle-clonus and dragging of the toes. The wasted muscles show fibrillary twitchings and give the reactions of degeneration. Contractures then occur, the forearms are flexed on the arms, the hands are held in pronation, and the proximal phalanges of the fingers bent backward, giving rise to the so-called *claw-hand*. From time to time there are tonic spasms in the muscles, particularly in the calves (*spinal tetanus*). Sensation is not disturbed, excepting for the occurrence of slight paresthesia from time to time, and the sphincters continue to functionate normally. Finally the bulbar symptoms appear, and there is paralysis of the lower part of the face, which becomes rigid and expressionless, with the mouth partly open and saliva dribbling from the angles. Deglutition and articulation become difficult or impossible, and death finally occurs from exhaustion or inspiration-pneumonia. During the course of the disease the intellect is slightly involved. Memory is impaired, the conduct becomes childish, and there is a tendency to weep or laugh without cause. Atypical cases occur in which either the lower extremities are first involved, or the paralytic symptoms are more prominent than the spastic symptoms, or the bulbar symptoms appear very early. The course is steadily progressive, and death usually occurs within two years.

The **differential diagnosis** is to be made from *multiple sclerosis* by the absence of nystagmus, of the intention-tremor, and of sensory disturbances, and by the degenerative changes in the muscles; from *transverse myelitis* by the absence of sphincter disturbance and of pain, and the involvement of the upper extremities and head; from *progressive spinal muscular atrophy* by the presence of spastic symptoms; from *syringomyelia* by the absence of sensory disturbances, trophic lesions of the skin and joints, and the greater regularity of the course; from *pressure upon the spinal cord* by the absence of pain and sphincter disturbance and the involvement of the head and upper extremities. It must be remembered that amyotrophic lateral sclerosis may be associated with multiple sclerosis or infantile spinal paralysis.

**Prognosis.**—It will be understood from the foregoing description that death is the invariable termination. The course is progressive, and even temporary amelioration rarely occurs.

**Treatment.**—The patient should be rendered as comfortable as possible. Arsenic and mercury are useless, but the hypodermic injection of strychnin (see Progressive Spinal Muscular Atrophy) may be tried.

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## SYRINGOMYELIA.

**Definition.**—A neuroglial overgrowth of more or less vertical extent, and situated in the gray matter of the cord in the neighborhood of the central canal. Its symptomatology is not constant, but the following have come to be looked upon as typical of most cases: viz. progressive muscular atrophy and dissociated anesthesia (*i. e.* impairment or loss of temperature—and pain-sense, with retention of the tactile and muscular sense and trophic and vaso-motor disturbances).

**Pathology and Etiology.**—Tubular cavities of greater or less extent are met with in the cord as a result of two conditions existing separately or in conjunction—viz. (1) *hydromyelia*, a dilatation of the central canal (proved by the cubical cells lining it). This is either (*a*) congenital, according to Leyden, or (*b*) acquired, due to pressure (tumor), dilatation taking place above the point of obliteration. (2) *Syringomyelia*, a name given by Olliver to a neuroglial overgrowth situated within the gray matter of the cord. In this cavity-formation takes place as a result of hemorrhage or degeneration. The cavity is entirely without the central canal; it never possesses an epithelial lining, and is not, therefore, as Leyden supposed, the remains of congenital hydromyelia. While the new growth in many instances is gliomatous, being probably a rejuvenescence of some vestigial remnant, with subsequent hemorrhage or degeneration and cavity-formation, yet in others the structure is not identical with such neoplasms. The latter have been described particularly by Joffroy and Achard. They speak of it as a gliosis, a secondary overgrowth, and sclerosis of the neuroglia. In any case, however, the disease is most prone to develop in the cervical and upper dorsal region, growing and invading the posterior and posterolateral tracts. Breaks and crevices in the diseased material radiate from the main cavity. The onset of the trouble generally takes place somewhere between the fourteenth and twenty-first years of age.

**Symptoms.**—Owing to the fact that different levels of the cord are involved, and that the extent claimed by the process varies in different cases, it will readily be understood that no account, however concise, will fit every case. The disease is of *slow onset*. Neuralgic pains develop in the muscles, and the latter progressively *waste*. The *reflexes* are increased and more or less spasticity is present. The lower extremities usually escape, though they too may be involved, when the condition presents much the same appearance as amyotrophic lateral sclerosis. The *temperature-* and *pain-sense* are lost, but the *tactile* and *muscular* senses are preserved.



The *special senses* and the sphincters are normal. *Ocular symptoms* develop only when the cervical cord is extensively involved. *Joint-changes* may be met with, and various *ulcerations, bullous eruptions, or wounds* may be present, the latter often being received without the patient's knowledge, since loss of sensation is complete. These constitute a special feature of a type of the disease originally described by Morvan of Brittany in 1883. He had observed many cases prior to that time, but his attention was specially called to the matter by a case of whitlow which he incised, but to his surprise no pain whatever was experienced. He described *the disease as affecting the upper extremities*, with neuralgia, progressive paresis and wasting, dissociated anesthesia, and, later, painless whitlows and necrosis of the phalanges. Joffroy and Achard have made three autopsies upon cases dying of this disease, and in each syringomyelia was found. In Gombault's case neuritis was present, and the current view is that *Morvan's disease* is a combination of syringomyelia and neuritis.

**Diagnosis.**—The loss of pain and thermic sense, with preservation of the muscular and tactile senses, in association with the muscular wasting, which is most marked in the upper extremities; and with the spasticity of the lower extremities, constitutes a group of symptoms that has come to be regarded as typical.

**Differential Diagnosis.**—*Hypertrophic cervical pachymeningitis* may be mistaken for this disease, and *vice versa*. In this case, however, the pain is usually greater, the tactile sense is apt to be lost, and possibly the other senses also; but there is not the dissociation met with in syringomyelia. *Amyotrophic lateral sclerosis* presents neither sensory nor trophic symptoms, other than the muscular wasting. *Disseminated sclerosis*, apart from the tremor that is always present, presents less trophic disturbance.

The **prognosis** is always unfavorable, though the disease runs a very chronic course, lasting even fifteen or twenty years.

**Treatment.**—Nothing can be done, except by attention to hygienic and dietetic details.

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## COMPRESSION OF THE SPINAL CORD.

It is of importance to be able to recognize this condition. To be sure, it is not always possible to diagnose it with certainty, but when there is a reasonably surety the question of operation may arise. Since it has so many features in common with myelitis, the necessity for reserve and caution in arriving at a conclusion is manifest, since the latter condition would not be benefited by any operative procedure.

**Pathology.**—The postmortem findings will depend upon the degree and duration of the pressure. The cord will be more or less flattened and distorted at the seat of pressure, and in the early stages hyperemic, and possibly softened. Later it is hard, sclerosed, and of a grayish color, and above and below the compressed region degenerated areas will be seen on sectioning the cord. Microscopic examination reveals

the same changes as those met with in any other form of myelitis. The nerve-roots will be more or less damaged by compression.

**Etiology.**—We may class the causes of compression under three headings—(a) traumatism (fractures), (b) inflammatory disease (caries of the spine), and (c) neoplasmata (including various tumors, gummata, and aneurysm), but these will receive separate consideration (*infra*).

**Symptoms.**—These will vary according to the site of the lesion and the extent of involvement—*i. e.* the vertical extent, the degree of pressure exerted, and the amount of inflammation present. Two groups of symptoms are present in typical cases—first, those due to involvement of the roots, and, second, those dependent upon involvement of the cord itself—*ascending* and *descending* degeneration. The former gives rise to *pain*, neuralgic in character and radiating along the course of the nerves. The parts supplied are usually *tender*, and there may be *paresthesia* and *formication*. These irritative symptoms are followed sooner or later by those of *paralysis*, and hence the anesthesia. Areas of *hyperesthesia* may accompany the anesthesia (*anesthesia dolorosa*). *Motor symptoms* are also irritative and paralytic, and hence the early twitching, or even spastic condition, and later the loss of power, or paralysis. The muscles of the affected parts waste, and qualitative and quantitative electric changes can be elicited. The *second group*, due to cord-changes, then develops, and its symptoms may set in either rapidly or slowly. If myelitis promptly supervenes and is extensive, *cord-symptoms* of a pronounced type develop quickly. The parts below the lesion will become weak, there will be girdle pains, and a sense of constriction or pain in the legs. Sensory symptoms may be absent. The reflexes are usually increased. If the tumor or other cause of compression ceases to fail to act for a time, some improvement takes place, due possibly to the subsidence of the myelitis. If the pressure is of *slow onset*, great tolerance is manifested. As is usually the case, sensation is recovered before motion. In certain cases, however, motor power is regained, while the muscular and tactile senses do not return. In such instances, in which the posterior columns bear the brunt of the trouble, incoördination results and secondary ataxia is met with.

**Diagnosis.**—If the combined symptoms of peripheral and central origin develop slowly in the order named, compression is likely. *Myelitis* gives rise first to cord-, and only later to peripheral symptoms; hence the difficulty in cases in which myelitis develops quickly. Extensive root-symptoms are suggestive of *meningeal involvement*. In any event, too much stress should not be placed on the nervous symptoms alone. The spine should be carefully examined and palpated for points of tenderness. Careful note should also be taken as to whether there is any limitation of movement or deformity. The family history may suggest *tuberculosis* (caries of the spine).

The **prognosis** depends entirely upon the cause. Having ascertained this, it then depends upon the possibility of its removal.

**Treatment.**—In general the treatment is that of myelitis. In certain cases a surgeon should be consulted, though operative cases are the exception rather than the rule, and most may be expected in cases of caries. It is well to impress upon the patient and relatives the chronicity of the condition, but faithful and persistent efforts will yield good

results. Rest is of vital importance, particularly when the disease is active. The patient should be kept in bed in a recumbent position until consolidation has taken place. Extension may be necessary. Good and easily assimilable food, and cod-liver oil and alteratives should be given. The nutrition of the muscles may be improved by gentle friction (massage). As soon as possible a plaster jacket should be put on the patient, and he should be taken into the open air and sunlight.

## TUMORS OF THE SPINAL CORD AND ITS MEMBRANES.

A GREAT variety of neoplastic formations, both primary and secondary in nature, may occur. The location is of course the most important clinical feature; nevertheless, if we would institute successful medical or surgical treatment it is desirable to determine as nearly as possible the nature of the tumor.

*Extradural tumors* may be, though rarely, exostoses from the periosteal lining of the spinal canal. More frequently lipomata or even an abnormal development of fatty tissue between the bone and the membrane is present. Of the parasitic growths, the hydatid cyst is apparently the only one that occurs in this situation.

The *tumors of the dura mater* itself are chiefly sarcoma, gumma, and myxoma; a rare form consists of the presence of plates of bone along the whole length of the cord. Occasionally growths from the bones, particularly carcinomata and sarcomata, may extend to the membranes. Inside the dural sac both hydatid cysts and the cysticercus may be found. Tuberculosis may occur either in the form of miliary tubercles or as tuberculous masses, particularly in the cervical region. In the pia mater and arachnoid, myxoma, lipoma, fibroma, endothelioma, sarcoma, and angio-sarcoma occur. Occasionally cylindroma and osteoma are also found. A specimen with bony plates in the dura was obtained from an old woman who had syphilitic lesions in other parts of her body.

In the cord itself the commonest tumors are glioma and sarcoma. Gummata also occur (*vide* Syphilis, p. 327). Cysts are sometimes found. The simplest form is the dilatation of the central canal known as hydro-myelia; the commonest are those that occur as a result of gliomatous softening in syringomyelia. Finally multiple neuromata may occur upon the nerve-roots. The changes that take place in the cord are destruction of the nervous tissue at the side of the tumor, pressure-myelitis, and systemic degenerations in the various columns. Curiously enough, complete restoration of function can occur, even when the symptoms of degeneration in the pyramidal columns are pronounced, and there is wasting of the cord macroscopically, as in the case recorded by Gowers and Horsley.

The **etiology** of these conditions varies, of course, with the nature of the growths. Certain tumors, as lipoma and glioma, are more apt to occur in advanced life; others, as the tyroma, somewhat earlier.

The **symptoms** depend upon the particular segment or segments affected, and the situation in the cord itself or its membranes. In



general the dorsal or lower cervical region is most frequently involved.

*Disturbances of Motion.*—If the tumor attains sufficient size to exert considerable pressure, paraplegia always occurs. This usually commences upon one side, and then more or less rapidly involves the other. *Exaggeration* of the *tendon reflexes* of the leg is almost invariable in tumors above the first lumbar segment. Whenever this condition is at all advanced there are disturbances of the functions of the bladder and rectum. *Spasms* are sometimes the earliest motor changes. Ordinarily they appear in the muscles of the trunk governed by the segment that has been involved; but sometimes they appear in the legs, and are usually more severe in one than in the other, and they may be due either to pressure upon the motor roots or to pressure upon the motor columns of the cord. It is not therefore permissible to draw definite conclusions from their location as to the site of the tumor. *Paresis* is commonly an associated symptom, and gradually deepens into paraplegia. In the latter stage *contractures* may also develop.

*Special Motor Symptoms.*—If the tumor is situated in the lower portion of the cervical region, there are often disturbances of *motility* in a certain definite group of muscles that are supplied by the brachial plexus. These disturbances may be *ataxia*, *tremor*, *spasm*, or *paralysis*. If the tumor be in the dorsal region, the cramp of the intercostal or abdominal muscles at a particular level may give rise to a *girdle-sensation*; if in the lumbar region, to disturbances of motion similar in character to those described in the arm. In this locality, however, as the nerve-roots are longer, more of them are apt to be involved in the pressure, and the symptoms are more extensive.

*Sensory Symptoms.*—*Pain* is usually the earliest symptom. It is, as a rule, sharply localized, severe, and paroxysmal, with symptoms of dull neuralgic aching between the exacerbations. Certain forms of pain are very common. Tumor in the cervical region gives rise to severe pain in one or both arms and to neuralgic pains in the neck and occiput. Tumors in the dorsal region cause the girdle-sensations before described, or intense backache, often associated with tenderness over the site of the tumor. Tumors in the lumbar region induce pain in one or both legs, often of a burning character, and sometimes referred to the soles of the feet. *Hyperesthesia* of the skin is usually found at a level supplied by the segment in which the tumor is situated. Other sensory disturbances are tingling, numbness, and total anesthesia. A rare combination, in the early stages of the tumor of the spinal cord, is the presence of anesthesia in one-half the body below the tumor and of paresis or paralysis in the other half—that is, the *symptom-complex of Brown-Séquard*: it almost invariably disappears in a short time. The reflexes are increased below the level of the tumor. If this is situated in the lower cervical region, then the cutaneous reflexes of the thorax and abdomen are prompt and vigorous; if in the dorsal region, they are normal above the site of the tumor, abolished in its neighborhood, and increased below, and at the same time there is an enormous exaggeration of the skin- and tendon-reflexes of the legs; if in the upper portion of the lumbar region, there may be an abolition of the knee-jerk, while ankle-clonus is exaggerated; but ordinarily all reflexes are abolished.

The muscles frequently degenerate, and the electric reactions of degeneration are found in those regions that are supplied by the anterior horn, or the anterior roots that have been destroyed by the tumor. In those tumors situated in the lumbar region, involving a number of nerve-roots, the wasting of the muscles of the legs is usually very marked. *Trophic disturbances* occur late in the course of the disease, when extensive bed-sores may develop, exactly as in transverse myelitis. Occasionally vaso-motor disturbances (*tâche spinale*, localized edema) may be observed in the early stages.

**Course.**—Tumors usually grow slowly, and therefore the symptoms are gradual in their development. Ordinarily there are periods of arrest or even improvement that are followed subsequently by further advance. The *duration* of spinal tumors is variable. Those of malignant nature or rapid growth may produce death in a short time; those that simply exert pressure and enlarge very slowly may not produce total disability for several years. In general it may be said that from five to ten years is the ordinary limit after the first appearance of motor disturbance. Some tumors, however, particularly lipomata, produce only slight disturbances throughout life, or else no symptoms at all, remaining entirely latent.

The **diagnosis** involves three points: first, the recognition of the presence of the tumor; second, of its site; and third, of its nature. The prodromal symptoms of spinal tumor are often confounded with *neuralgia* or *lumbago*. It is sometimes possible to make a **differential diagnosis** by means of the presence, in neuralgic conditions, especially of intercostal nature, of the sensitive points along the course of the ribs, and of the existence, in the case of tumor, of exaggerated knee-jerks and sensitiveness over certain portions of the vertebral column. In the paraplegic condition it may be confounded with a *neuritis*, such as one of alcoholic origin. In these cases the diagnosis is more difficult if the tumor is situated in the upper portion of the lumbar cord; nevertheless, the sensory disturbance is slight in alcoholic neuritis, whilst it is considerable in tumors in the lumbar region, and often presents the form of *anæsthesia dolorosa*, that is, diminished sensibility associated with considerable pain. There are also apt to be disturbances of the sphincters.

The *intrinsic diseases* of the spinal canal give rise to much greater difficulty, especially *myelitis* and *pachymeningitis cervicalis*. From the former the correct diagnosis may sometimes be suspected, because in tumor there are severe radiating pains and the symptoms are more pronounced on one side than on the other. Moreover, the symptoms of segmentary involvement are sharper and the root-symptoms more characteristic. From *pachymeningitis cervicalis*, a central tumor in the cervical region can be usually distinguished by the fact that the radiating pains are less severe and the symptoms not so distinctly bilateral. It may be impossible to distinguish a central tumor from *syringomyelia* unless the symptoms of root-pressure are quite distinct. *Pott's disease*, in its early stage, may also give rise to some difficulty. However, the rapid development of the kyphosis, and particularly the pain that is elicited by sudden pressure upon the head, renders it possible, after a reasonable period of observation, to recognize the true nature of the case.

The *diagnosis of the position of the tumor* has been largely discussed

in the Symptomatology. The symptom-complex may, however, be considerably disturbed by the presence of multiple tumors. In these cases the majority ordinarily remain latent. It must not be forgotten, however, that the absence of the knee-jerk does not localize the tumor to the lumbar region of the cord, for it may be abolished when the tumor is situated in the dorsal region and compresses the posterior columns. In general, it may be said that the presence of root-pains suggests a meningeal seat, whilst pronounced paraplegia, or the Brown-Séquard symptom-complex, points to the presence of a tumor in the substance of the cord itself.

Finally the recognition of the *nature of the growth* can often be made from the history of the existence of a tumor or an infectious process in other parts of the body. It must be remembered, however, that it does not always follow that a tumor in the spinal canal is similar to that found elsewhere. The presence of cerebral as well as spinal focal lesions points very strongly to syphilis.

The **prognosis** depends upon the severity of the symptoms, the rapidity of their development, and the nature of the growth, if this should be known. Complete subsidence of all the symptoms may occur, even after a spastic paraplegia has existed. Of course this is only likely in those cases in which the tumor can be removed by operation or absorbed through the action of drugs.

The **treatment** depends wholly upon the recognition of the nature of the tumor; if this be syphilitic, mercury and potassium iodid should be given in full doses. If, on the other hand, it is not specific, and appears to be extradural, operation would seem to offer a possibility of cure, the famous case of Gowers and Horsley having demonstrated the practicability of removal. As the prognosis is, in general, unfavorable as to cure and often gloomy as to life, the clinician should not hesitate to recommend surgical interference.

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## LESIONS OF THE CONUS TERMINALIS AND THE CAUDA EQUINA.

As the spinal cord terminates at the second lumbar vertebra, tumors or injuries below this point produce symptoms only in so far as they compress or destroy the lumbar roots. This destruction may be partial or complete. If partial, there are *paralyses* of various groups of muscles, and circumscribed areas of anesthesia. There may or may not be a disturbance of the functions of the bladder and sphincters. If this be *total*, there are complete anesthesia, complete paraplegia, flaccid in character, with reactions of degeneration in the muscles, loss of the knee-jerk, and rectal and vesical incontinence. If only the *cauda equina* is involved, there may be isolated paralysis of the bladder and rectum. These lesions may consist of tumors, such as are found in the membranes of the cord or on the nerve-roots, and it should be noted that, probably on account of greater space for their development, tumors in this situation are apt to be larger than those in other parts of the spinal canal. They may also consist of fractures or lesions occurring as a result of congenital anomalies, such as spina bifida.



## III. DISEASES OF THE BRAIN AND ITS MENINGES.

## DISEASES OF THE DURA MATER.

**Inflammation.**—This may be met with on the outer or inner surface (*pachymeningitis externa* or *interna*). Of the external variety the chief causes are (*a*) traumatism, (*b*) disease of the bone, (*c*) syphilis, and (*d*) middle-ear disease. That due to traumatism is often seen, and in the mildest form is of little moment. When severe and accompanied by fracture with or without displacement, infection of the membranes may either take place at once or later from diseased bone. That form due to caries or any other form of osteitis is always dangerous, owing to the possibility of infection of the diploë. The brain-sinuses will then become affected, and infected emboli may pass into the circulation, with the development of pyemia. In the syphilitic variety the inner table of the skull is thickened and roughened, and more or less pus and granular material is found between it and the dura (see also Syphilis of the Nervous System). Sinuses may communicate with the exterior.

The **symptoms** are indefinite in mild cases, and may consist only of *headache*. In the severe forms there are *headache*, *malaise*, *chills*, *fever*, *drowsiness*, and later *stupor*, and rarely *convulsions* and *hemorrhage*, or other symptoms of compression. The ophthalmoscope will reveal more or less evidence of *choked disc*. Rigors are suggestive of the onset of pyemia.

The **treatment** varies with the cause. Antiphlogistic measures and counter-irritation are of value, and in the severe grades operative interference may be necessary. The internal variety either occurs as a simple inflammation or may be so acute as to cause extravasation of blood. This may organize, and, together with the products of inflammation, cause a pseudo-membrane. Rarely is pus found.

**Hemorrhage.**—Hemorrhage may be (1) extradural—(*a*) traumatic and (*b*) due to rupture of a vessel by erosion, the result of caries; or (2) intradural—into the so-called arachnoid sac—(*a*) very rarely traumatic; (*b*) due to injuries at birth; (*c*) due to pachymeningitis interna; (*d*) met with in general paralysis of the insane; (*e*) occurring in the course of anemia, scurvy, or some other profoundly altered blood-condition; (*f*) in cardiac, renal, or pulmonary disease; (*g*) the result of strain—*e. g.* whooping-cough.

The **symptoms** will depend upon the circumstances, whether the amount of blood is small or large, whether the onset is *gradual* or *abrupt*; they may be further obscured by the primary disease, or by *shock* if the cause is some trauma. In the slight forms absolutely nothing characteristic exists. In others there are *headache*, *vertigo*, *vomiting*, and possibly *mental confusion*, *convulsions*, or *coma*; in fact, the ordinary symptoms of apoplexy.

The **treatment** is that of cerebral hemorrhage; in some cases operation is justifiable.

**Internal hemorrhagic pachymeningitis** or hematoma of the dura mater is characterized by the formation of a fibrous exudate upon the inner surface of the dura, into which capillaries extend that subse-

quently rupture. It is found most commonly among the insane or epileptic. It is rare in childhood.

The **symptoms** are variable. The entire course may be without symptoms, or they may be masked by the existence of other conditions. More frequently there are *headache* and *convulsions*, followed later by *paralyses*, coma, and death. The location of the lesion causes considerable modification of the symptomatology. In the milder form recovery frequently occurs, or the case may become chronic. If the onset is sudden, the symptoms may resemble those of hemorrhage.

The **diagnosis** is always difficult. In children muscular contractions and convulsions are frequently met with; in adults the slow onset may be the only difference between this condition and an attack of *grand mal*. Of course, there is a greater periodicity in epilepsy; but a repetition of the attacks occurs in hematoma, and, as already stated, the repeated hemorrhages are believed by some to be the cause of the lamination of the false membrane.

The **prognosis** is extremely unfavorable in children, but is much less so in adults.

The **treatment** calls for the use of leeches behind the ears and over the temples, the ice-cap, and counter-irritation. Free movement of the bowels should be promptly secured, and later the iodids or mercurials should be administered.

**Tumors** are considered in connection with Tumors of the Brain.

## DISEASES OF THE PIA.

**Inflammation** (*Leptomeningitis*).—This is met with in the following conditions: (*a*) When tubercles develop on the membrane (*vide* Tuberculous Meningitis); (*b*) During, or as a sequel to, some acute febrile disease, as pneumonia, erysipelas of the head and face, small-pox, measles, scarlet fever, typhoid fever, ulcerative endocarditis, and pyemia; (*c*) Cerebro-spinal meningitis; (*d*) Cachexia; (*e*) Gout and Bright's disease; (*f*) Exposure to the sun; (*g*) Traumatism, even when not accompanied by fracture; (*h*) Disease of the bones—caries, or secondary to middle-ear disease; (*i*) Extension from syphilitic involvement of the meninges.

Inflammation of the pia (non-tuberculous) is more common in males than females, and occurs, as a rule, before the twentieth year. After that time it is rather rare.

**Pathology.**—In the extent and degree of the inflammation, great variations exist. It may be either (1) limited to the convexity, with or without involvement of the sides; (2) limited to the base; or (3) general, involving both convexity and base. In the early stages and in the mild forms there may be no more than an injection of the part. Later, inflammatory products are met with and more or less adhesion exists. Pus is also present in some cases. This form of leptomenigitis, unlike the tuberculous variety, is prone to attack the convexity of the brain. I have made an autopsy on one child of twenty months in which the in-

inflammation was slight on the convexity, and still less on the sides, but quite marked on the base, particularly about the Sylvian and cerebellar regions and the optic chiasm. A granular ependymitis was found, limited to the body of the lateral ventricles, and reminding one very much of a similar condition met with in paralysis of the insane. The ventricles were dilated, and contained an excess of fluid, due probably to occlusion of the foramen of Magendie, as Gee and Barlow have pointed out. No tubercles were found, nor did the family history lead me to suspect tuberculosis.

**Symptoms.**—These are very varied, and naturally depend on the *seat* and *extent* of the inflammation. Those cases in which symptoms pointing to involvement of the base occur need not be discussed here, since they are considered in detail under the tuberculous variety. In any case *headache*, localized or general, is usually present. In children too young to talk its presence is often indicated by crying or putting the hand to the head. *Delirium*, *insomnia*, and *coma* are also met with in different cases. There are more or less *fever*, *constipation*, a *coated tongue*, *vomiting*, a *rapid pulse*, and the *tâche cérébrale* may be elicited. Spasmodic movements may occur, or even general *convulsions*. Of course, in cases of inflammation of the base, the cranial nerves become affected, and we have *ptosis* or *strabismus*, *facial spasm* or *palsy*, and, if the fifth nerve is involved, *sensory* and *trophic* changes. It must be borne in mind that meningeal symptoms are frequently simulated by the *infectious diseases*, and particularly by pneumonia (of the apical type generally; more especially in children and old people), typhoid fever, and influenza. Such cases are most likely due either to vascular disturbance of the meninges (congestion) or to a toxic encephalopathy.

**Diagnosis.**—Where no etiologic hint can be obtained the diagnosis is generally in doubt for two or three days. There may be nothing more than a reflex irritation (dental or gastro-intestinal), or possibly one of the infectious fevers. The symptoms should be studied in their entirety; one or two supposedly pathognomonic signs should not be allowed to cloud our vision. Having made the diagnosis of meningitis, it becomes important to **differentiate** the *tuberculous* from the *non-tuberculous* variety. The family history is of importance. In *tuberculous meningitis* the focal symptoms usually appear late, and are due to involvement of the cranial nerves at the base of the brain, chiefly those controlling the eye. The eye-grounds often show a slight perineuritis without choked discs, and perhaps one or more miliary tubercles. The leukocytes are slightly, if at all increased. There is rarely rigidity of the neck. In other forms of meningitis this appears early. The optic nerve shows intense inflammation and there is usually pronounced leukocytosis. Quinke, in 1890, introduced his method of *lumbar puncture*. This is of comparatively simple application, and has become a well-recognized measure. The tubercle bacillus and streptococcus pyogenes, also the pneumococcus and micrococcus meningitidis, have frequently been found in the fluid withdrawn.

**Prognosis.**—This is always grave. A percentage of cases of epidemic cerebrospinal meningitis, varying with the nature of the epidemic, may recover. In all other forms any termination, except in



death, is exceedingly exceptional. Remissions frequently occur in the symptoms, and the course may be very prolonged. That even tuberculous cases may recover is proved, however, by that of West, and even more strikingly by that of Baumann and Senlie, who found the tubercle bacillus in the cerebro-spinal fluid. The case recovered, but died some months later from an intercurrent affection; the diagnosis gained additional confirmation from the *postmortem* findings.

**Treatment.**—This has been previously considered. It is only necessary to add that we are able to do no more in this form than in the tuberculous. We have no specific, and all that can be done is to meet the symptomatic indications. In certain cases—*e. g.* those secondary to middle-ear disease—operation may seem justifiable. When in doubt the physician should not delay action until too late, but should call in a surgeon while some benefit may still be hoped for.

## DISTURBANCES OF CIRCULATION OF THE BRAIN.

### HYPEREMIA.

**Definition.**—An abnormal increase in the amount of blood in the cerebral capillaries. The condition is not in any way associated with the primary phenomena of inflammation.

What has already been mentioned in the case of hyperemia of the cord is equally true in this case—*viz.* that while congestion undoubtedly may take place, there is nothing symptomatically pathognomonic in the fact, and hence we do not recognize it as a definite clinical entity. "Congestion of the brain" is rather a "diagnostic haven" and satisfies the patient, while at the same time, provided the assumption is not made on too superficial evidence, it harms no one. Two forms of hyperemia have been described:

1. **Active hyperemia** is met with in men more frequently than in women, and results from over-action of the heart and widespread obstruction to the circulation, as when the surface capillaries contract, or there is arterial dilatation, due to excessive mental activity from any cause or to drugs—alcohol, amyl nitrite, nitroglycerin.

2. **Passive congestion** is met with in cases of obstruction of the cerebral sinuses and veins, and is due to pressure on the superior vena cava or the innominate or jugular veins by tumors or aneurysms; also in suffocation and strangling, in cases of excessive strain, and in triepid insufficiency.

**Pathology.**—There are no marked changes in the brain in these cases. In the active form the gray matter will appear somewhat darker than normal, and a macroscopic section of the white matter shows the puncta vasculosa to be increased. It often leaves no trace *postmortem*. In passive congestion the veins and sinuses are engorged and more or less edema may be present.

The **symptoms** are described under two headings—1, those of *irritation*, and, 2, of *depression*. Among the former are *headache, vertigo,*

*irritability, rapid pulse, restlessness, insomnia*, and special nervous phenomena, as *flashes of light, hyperacusis*, and even *convulsive movements*. The latter is manifested by the obtunding of the senses; in fact, the antithesis of the other. The cerebral symptoms met with in febrile processes are probably due either to faulty metabolism or to some toxin, and should not be regarded as the result of hyperemia.

**Treatment.**—The recumbent posture is of great importance. Leeching, wet-cupping, and venesection are sometimes employed. Cold applications to the head, bromids internally, and attention to the bowels will be of assistance. Freedom from annoyance and worry of all kinds is necessary.

#### ANEMIA.

**Definition.**—A condition in which an insufficient amount of blood circulates in the cerebral capillaries.

It is due to exhausting discharges (diarrhea), an abnormally slow pulse or weak heart, to hemorrhage, obstructive endarteritis of the vessels supplying the brain, and to syncopal attacks and dilatation of the intestinal vessels, owing to the too rapid withdrawal of ascitic fluid.

**Pathology.**—The gray matter is quite pale; the *puncta vasculosa* are diminished, and sometimes cannot be seen; the cerebro-spinal fluid is frequently increased.

**Symptoms.**—The most exaggerated type is met with after a profuse hemorrhage. There are *pallor, weakness, vertigo, headache, flashes of light, subjective noises, rapid respiration, cool skin*, possibly profuse sweating, and in extreme cases *coma, convulsions*, and *death*. We are more familiar with the ordinary fainting-attack. When cerebral anemia is brought about more slowly “irritable weakness” results. The patient is either *somnolent, dull, and apathetic*; or he may be a victim of *insomnia*. *Headache, vertigo, tinnitus aurium, musca volitantes*, and lowered muscular power are present. The patient becomes irritable on the slightest provocation. Marshall Hall has described a group of symptoms as “hydrocephaloid” from their resemblance to hydrocephalus; they occur especially in young children after diarrhea. There are *pallor, hebetude, contracted pupils*, and *depressed fontanels*. The somnolence may deepen into a coma that often becomes more profound until death results.

The **treatment** varies with the cause. The recumbent posture is always indicated, and in some cases it is necessary to depress the head, administer stimulants, and even transfuse or inject a normal saline solution. A light and easily assimilable diet should be given during convalescence.

#### EDEMA OF THE BRAIN.

**Definition.**—An infiltration of serum into the subarachnoid space and a greater or less increase of ventricular fluid, with or without infiltration into the brain-substance.

**Pathology.**—The fluid is chiefly in the meshes and beneath the membrane. The ventricular fluid is increased in amount; the brain-substance is pale, and in some cases infiltrated and softened. Microscopically, *lacunæ* may be seen in the cerebral tissue, the perivascular

spaces are dilated, and some slight degree of nerve-cell degeneration is often present.

**Etiology.**—Edema is met with in Bright's disease, in senile cerebral atrophy, and as a result of active or passive hyperemia.

**Symptoms.**—In general the symptoms are those of *anemia* though nothing definite is known of them. Since the condition is always secondary, it may be that symptoms directly referable to the edema are masked by the primary condition. Cases of apoplexy are seen occasionally, in which the only postmortem finding is an effusion of fluid into the pia and ventricles. This has been termed "serous apoplexy."

The **treatment** is that of the primary condition.

#### EMBOLISM AND THROMBOSIS.

(*Cerebral Softening.*)

**Embolism.**—**Definition and Etiology.**—The obstruction of arteries or capillaries by material brought to the spot from some other part by the blood-current. The material, generally fibrin, usually comes from the heart, and is either a vegetation of a recent endocarditis or, more commonly, of chronic valvular disease; it may possibly be a fragment of the valve plus the fibrin in ulcerative endocarditis. In the latter case the plug is generally septic, giving rise to suppurative processes. An embolus may be washed from the auricular recesses, from an aneurysm of the aorta or carotid, or from atheromatous patches; rarely from the pulmonary veins.

In puerperal women, and in certain febrile processes (diphtheria and pneumonia) the coagulability of the blood is increased. Heart-clots form, and fragments may be washed into the cerebral vessels. Owing to the direction of the vessels the embolus most frequently enters the left carotid, whence it usually passes to the left middle cerebral. Almost any cerebral artery may be obstructed, but the cerebellar very rarely. Embolism occurs most frequently between the tenth and fortieth years of life. The middle cerebrals are most frequently involved, and next in order the internal carotid and anterior cerebrals.

**Pathology.**—That region of the brain that is cut off from its blood-supply by the embolus undergoes softening. The cortical changes are less marked than those of the central ganglia, since in the former case more or less anastomosis exists, and none in the latter. When the embolus is septic one or more metastatic abscesses result. The degree of softening varies in different cases within wide limits. There may be nothing more than a slight diminution in the consistence, the affected area being somewhat paler than normal, or absolute dissolution may occur, the myelin breaking up into granules, while the tissue becomes infiltrated with serum, and the vessels undergo hyaline or more often fatty change. The color of the part varies with the amount of blood. In recent cases it is red. As the hemoglobin is absorbed a yellow color appears, and soon predominates. Red and yellow softening are found chiefly in the cortex. The so-called white softening is met with particularly in the white matter. A variety of red softening in which numerous small hemorrhages exist has been termed capillary apoplexy, while



*plaques jaunes* is the term given by the French to a form of yellow softening often seen in the cortex of old people. The ultimate changes depend in a great measure upon the extent of the lesion. If this is small, the granular débris is absorbed, and the proliferation of connective tissue results in the formation of a scar. On the other hand, if large the solid elements are removed, and the cavity that remains contains more or less fluid (a cyst). In many instances fibers, trabeculæ, and even vessels that have escaped destruction, pass through the cyst.

**Thrombosis.**—**Definition.**—Obstruction of a vessel due to clotting *in situ*. This may occur (a) in the arteries or (b) in the veins and sinuses.

**IN THE ARTERIES.**—**Etiology.**—Thrombosis results from disease of the vessel-wall, atheroma, endarteritis, or syphilitic arteritis, extension from surrounding diseased areas, traumatism, in aneurysms, in depraved blood-states, and at the seat of lodgement of an embolus. Thrombosis of a cerebral vessel may rarely follow ligation of the carotid. In general we may say thrombosis results from (1) changes in the vessel-wall, (2) retardation of the blood-current, and (3) hypercoagulability of the blood. It occurs most frequently in the middle cerebral, basilar, internal carotid, and vertebral arteries.

**Pathology.**—The changes in the brain-tissue are precisely those described under Embolism. Within the vessel a clot is found adherent to the vessel-wall, and extending from the nearest large branch on the proximal side to the contracted branches on the distal side. If of recent and rapid formation, it is always of a red color. The slower the formation the paler the color. Such clots are often laminated. The ultimate changes are contraction and atrophy, or, more rarely, calcification, or even softening and removal, the vessel again becoming patulous.

**IN THE VEINS AND SINUSES.**—**Etiology.**—Thrombosis may be (1) primary, due to general causes, or (2) the result of local changes.

*Primary thrombosis* is less common than the secondary variety. It is met with in marasmic children (one of the causes of infantile hemiplegia—Gowers), in which the clot is called marantic thrombosis, cachexia, phthisis, carcinoma, and in blood-dyscrasiæ (anemia, chlorosis).

*Secondary thrombosis* usually results from an extension of neighboring forms of inflammation, caries of the bone, middle-ear disease, or meningitis. It may also be due to fracture of the skull or compression of a sinus by a tumor.

**Pathology.**—In primary thrombosis the most common seat is the superior longitudinal sinus. From this it spreads into the veins of both sides, and frequently also into the lateral sinuses of one or both sides. In secondary thrombosis the sinus nearest the local disease suffers. The veins emptying into the sinus involved become distended, often rupture, and in consequence the brain-tissue and pia become infiltrated. When the veins of Galen are blocked serum escapes into the ventricles. Red, yellow, and white softening is met with as a final result of the extravasation. Secondary thrombi are usually septic. Primary thrombosis is probably due in many cases to an excess of carbonic acid in the blood. Chlorotic blood is probably charged with it. The comparative infrequency, however, of clotting in the cerebral sinuses is

due to the fact, recently shown, that they contain very little carbonic acid.

**Symptoms.**—**Following Embolism or Thrombosis of Arteries.**—The symptoms necessarily depend upon the position and extent of the lesion. Often it is discovered *postmortem*, not having been suspected during life. We meet with many such cases occurring in late adult life. Then, too, extensive lesions may occur in those portions of the brain that never yield any localizing symptoms—the frontal region, for instance. Apart from the etiologic differences, the clinical pictures of embolism and thrombosis differ as follows: In the former the *onset* is sudden, without premonitory signs, and is in many cases accompanied by loss of consciousness. In addition to symptoms arising directly through implication of the particular part involved, there are those of *shock*. In the less severe cases consciousness soon returns and the apoplectic symptoms pass off. When more severe, *coma* supervenes and may prove fatal. When hyperemia occurs in or about the motor region the irritation may give rise to *convulsions*. In other cases *delirium* is a prominent feature; hence three varieties of softening are described by some writers—the *apoplectic*, *convulsive*, and *delirious*, from the prevailing feature. Thrombosis may commence abruptly, but as a rule the onset is slow, the patient meanwhile complaining of vague pains, numbness, tingling, headache, and vertigo. It is observed that a gradually increasing impairment of the mind is going on, and that motor weakness, slight at first, increases until the function is lost. The special symptoms are, as stated, dependent upon the location of the obstruction. If this is in the middle cerebral artery, the most common seat, there will be *hemiplegia*, owing to destruction of the internal capsule. The trunk may be spared and one of its branches stopped. The latter run to the third frontal, ascending parietal, supramarginal, angular, or temporal gyri. Thus, then, we can account for the aphasia so often met with in these cases by the plugging of the branch that supplies the third frontal convolution of the left side. If both middle cerebrals are plugged, symptoms develop that are indistinguishable from hemorrhage into the ventricles. This condition is generally fatal. Thrombotic obstruction of the anterior and posterior cerebral arteries rarely causes symptoms, owing to compensatory circulation. “Hebetude and dulness of intellect may occur” (Osler), with obstruction of the anterior cerebral. Hemianopsia may arise from a lesion of the posterior cerebral, since it supplies the cuneus. The left cerebral is more often involved than the right. In either case bulbar symptoms develop.

**Cerebellar softening** is rare. When it does occur it is usually in the region supplied by the posterior cerebral artery. There may be no symptoms if only one hemisphere is involved: otherwise they are similar to those of cerebellar disease due to other lesions.

**Thrombosis in veins and sinuses** causes variable symptoms. Those directly due to the vascular disturbance are severe headache, optic neuritis, delirium, or convulsions, and, later, great depression. Hemiplegia may result. When the superior longitudinal sinus is affected, epistaxis is common, while in lateral-sinus involvement post-auricular edema occurs. In secondary cases, moreover, we have to reckon with the cause. Since this is so often septic, septicemic symptoms are the rule.

**Treatment.**—*Of Embolism and Thrombosis of Arteries.*—Very little can be done in brain-softening. In the early stages, however, while it is absolutely impossible to repair the tissue already damaged, an effort should be made to prevent the spread of the process. Rest in bed with the head slightly elevated should be insisted on. When shock is present it must be met by gentle stimulation, ammonium carbonate, and even by alcohol and digitalis in some cases; hot-water bottles may be applied to the body. Venesection is contraindicated. The bowels should be made to move gently and purgation should be avoided. Later, as stated, symptoms of irritation often appear. In such cases the bromids should be given, and also a diaphoretic mixture, or ice should be placed to the head. In any case in which syphilis, rheumatism, gout, chorea, or other malady capable of causing or adding to the trouble exists, the original disease should be treated promptly and thoroughly. In the mean time efforts should be made to improve the patient's general tone by the strict observance of hygienic and dietetic rules.

*Of Thrombosis of Veins and Sinuses.*—Treatment in these cases depends largely on the cause. In the primary form it is that of the systemic disease. Good, wholesome, and easily assimilable food should be given, together with a tonic treatment. In secondary thrombosis careful search should be made for pent-up inflammatory products, which should be liberated at the earliest possible moment. The emunctories must act freely. Counter-irritation, applied to the neck, is of questionable value, but internally quinin, iron, and strychnin, and, if stimulation is necessary, ammonia and alcohol, will all be useful.

#### VASCULAR DEGENERATION.

**Arterial.**—The cerebral arteries undergo a more or less decided degenerative change in the majority of people past middle life (Bichat said seven-tenths). It is met with much earlier, however, as a result of disease. Bright's disease, rheumatism, gout, alcoholism—in fact, any irritation of the vessel-wall, whether autogenous, the result of faulty metabolism, or whether introduced from without, as alcohol—is capable of bringing about a change of the inner seat of the vessel, to which Virchow gave the name "*endarteritis deformans*." The circle of Willis and its branches are the most frequent seats. Various stages may be met with in different vessels or even in the same vessel—viz. hyaline degeneration, fatty degeneration, liquefaction-necrosis, atheromatous ulcers, and calcification.

Syphilitic arteritis is not a true degenerative process. It is rather a proliferative process in which both intima and adventitia are involved.

**Venous.**—The veins are less liable to disease than arteries, possibly because they are more yielding, yet the same pathologic changes may be met with in them. They are more commonly damaged by extension of inflammation from neighboring tissues or by pressure.

**Aneurysm.**—Dilatation of a vessel results from any of the causes above mentioned. The aneurysms may be very small—miliary—or often as large as a filbert-nut, and rarely as large as a hen's egg. They occur more commonly in males than in females. The middle cerebrals and basilar are most frequently attacked, and next come the internal



carotid, the vertebral, and the anterior and posterior cerebrals. Miliary aneurysms are frequently found in enormous numbers upon the basilar branches of the cerebral arteries.

*Symptoms of Aneurysm.*—There may be none; but in any case they are due to pressure exerted by the mass, and are therefore comparable to tumors of the brain. In many cases the first evidence of any trouble is an *apoplectic attack*, and it is scarcely necessary to add that this is usually fatal. In other cases *headache*, *vertigo*, and *optic neuritis* are present, and more rarely a *subjective murmur*. Still more rarely an *objective murmur* may exist.

## INFLAMMATION OF THE BRAIN.

(*Encephalitis.*)

**Definition.**—Encephalitis, strictly speaking, is an inflammation of the brain-substance, and does not include inflammation of the meninges, though in many instances the two conditions coexist as the result of a common cause, or one may precede and give rise to the other. Encephalitis is met with in two forms—(a) Focal, and (b) Diffuse.

### FOCAL ENCEPHALITIS.

(*Abscess.*)

**Pathology.**—In very acute cases no time is given for encapsulation; when of longer duration, however, the abscess is well circumscribed, having a well-defined wall, within which there are cell-detritus, pus, and sometimes more or less altered blood. It may be offensive. About it the brain-substance is generally softened and edematous. The abscess is generally single, except in pyemic cases, and varies greatly in size in different instances.

**Etiology.**—Abscess of the brain is a more or less circumscribed process, due to (1) *Injury*.—In the majority of cases of abscess following head-injuries either a compound fracture of the skull exists, with or without *hernia cerebri* (*fungus cerebri*), or a punctured wound has been made. Less commonly it may follow a simple fracture, and rarely it is said to occur when neither a fracture nor even an abrasion of the scalp has been produced. Meningitis is an almost invariable concomitant. (2) *Extension from some neighboring inflammatory focus*, as from orbital, nasal, or aural disease, in which the bones have usually become affected. (3) *Pyemia*, in which case the abscesses are apt to be small and multiple. It is also met with occasionally in gangrene of the lung, bronchiectasis, ulcerative endocarditis, suppurative hepatitis, or bone-disease, and rarely in chronic septic processes. (4) *Congenital Heart-disease*.—Little is known of this condition. Within the past two or three years Northrup, Packard, Sir Dyce Duckworth, and Osler have reported such cases. (5) *Obstruction of an artery, vein, or sinus*, whether of spontaneous origin or the result of ligature, may give rise to abscess. Generally, however, the cerebral change is that of softening, and not of true suppuration. (6) *Intracranial tumors*. (7) *Infectious fevers*.

**Symptoms.**—These at first are generally vague; but in traumatic cases, and more especially in those in which a compound fracture of the skull has resulted, the course may be most acute, and *fever, headache, delirium*, and possibly *vomiting* may be seen quite early. These are followed by other evidences of irritation, soon by compression with *convulsions*, and then by coma and death. In the more chronic cases the symptoms depend upon the size and location of the abscess and whether or not a vent exists. In such cases an intermission in the symptoms is occasionally met with, due to filling and emptying of the sac. Apart from the headache, pyrexia (not always present), twitching, and drowsiness, that occur in the course of these cases, more or less *hemiparesis* commonly exists, except in abscess of the frontal lobes. The latter are spoken of as “silent regions.” An abscess may be “latent,” however, in almost any region, these latent abscesses being typified in certain cases of congenital heart-disease. I do not think they were suspected during life in any of the cases reported thus far, and therefore optic neuritis has not been looked for; in other cases this latter symptom is commonly present.

**Diagnosis.**—In the acute cases following injury little difficulty presents as a rule, though even in this group a latent period may exist. With such a history, however, the onset of headache, fever, delirium, and convulsive movements is decidedly suspicious, and, should optic neuritis also exist, practically no doubt can remain. When dural or nasal disease exists the head-symptoms should be carefully studied, since they are prone to develop in ear-disease soon after a cessation in the discharge.

**Differential Diagnosis.**—Brain-tumor usually runs a more chronic course, and is seldom accompanied by fever, at least not until its final stage. It may be impossible to differentiate cerebral abscess from meningitis, and the two conditions often coexist, as already stated.

The **prognosis** is always grave.

**Treatment.**—When an abscess is diagnosed immediate operation is indicated. Suspected cases may be treated symptomatically unless focal symptoms develop. It must be remembered, however, that in a great many cases no localizing symptoms appear, and, since we know that most abscesses occur either in the temporo-sphenoidal lobe or in the cerebellum, when we have reason to suspect the presence of one, these regions should be explored.

#### DIFFUSE ENCEPHALITIS.

A good deal of doubt exists in the minds of some as to whether diffuse encephalitis ever exists except as a result of traumatism. Certain it is that it is less common and much less is known about it than of inflammation of the cord. We meet with it especially in the frontal regions in certain cases of general paralysis of the insane. The changes often escape the unaided eye. Microscopically, the vessels will be found injected and the perivascular spaces distended with leukocytes; these latter escape into the surrounding tissue, which becomes softened and edematous.

**Symptoms.**—It is manifestly impossible to give a definite symptomatology in the present state of our knowledge. Excepting the traumatic cases the symptoms are, as a rule, chiefly psychic.

**Acute hemorrhagic encephalitis** is a condition described by Strümpell. The brain and soft membranes are hyperemic and slightly edematous, and throughout the brain-substance there are numerous punctate hemorrhages. Perivascular leukocytic infiltration is also present. The *etiology* is unknown; but in some cases the disease appears to follow influenza. The *symptoms* are grave from the first. There are intense headache, fever, unconsciousness, disturbances of motility that are usually hemiplegic in type, and finally death. In less acute cases there may be delirium, rapid emaciation, and symptoms of involvement of the cranial nerves. The *diagnosis* can rarely be even suspected. The *prognosis* is invariably fatal, and no *treatment* is of any avail.

**Acute polioencephalitis**, so-called, is a disease of childhood of doubtful pathology. In some of the cases after recovery from the acute process insular sclerosis or porencephaly is present, with secondary degeneration in the pyramidal tracts. The *etiology* is obscure. The *symptoms* resemble those of acute poliomyelitis of the cord; but contractures develop very rapidly in the paralyzed limbs, and reactions of degeneration are absent from the muscles. The *prognosis* and *treatment* are the same as for the disease of the cord.

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## CEREBRAL HEMORRHAGE.

(*Cerebral Apoplexy.*)

**Definition.**—Hemorrhage into the brain-substance; though bleeding into the meninges, which rarely co-exists, is generally embraced in the definition.

**Pathology and Etiology.**—In intracerebral hemorrhage the blood will be found to have infiltrated the brain-substance, and, if extensive, it may have penetrated into the ventricle. In such cases the white matter is torn asunder, leaving a ragged space that is more or less filled with recent clot and fragmentary gray matter; if the ventricles have been entered, blood may escape from the lowest into the subarachnoid space. In less severe cases the territory involved is less extensive, and the blood may occupy a single space or several small spaces, forming mere separations of the nerve-fibers. Other changes take place according to the duration of the case. The blood changes color and gradually grows lighter, while reactive inflammation about the lesion results in the formation of a wall. The cyst—for such it has become through fatty degeneration of its contents—may remain as such, or when the lesion is a small one connective tissue may form within and a scar result. The larger arteries are generally atheromatous, and an aneurysm is occasionally met with; many miliary aneurysms may be seen in the course of the smaller vessels. It is very seldom that the actual source of the hemorrhage can be discovered.

Secondary degeneration follows a lesion occurring in the motor region (the cortex or internal capsule), so that sclerotic changes can be traced from the cortex through the corona radiata, internal capsule, crura, pons, and medulla to the termination of the fibers in the cord.

Cerebral hemorrhage is generally of arterial or capillary origin. It is



rarely venous, and in the latter case is due almost always either to traumatism or to rupture. Spontaneous rupture generally results from extension of some neighboring focus of disease to the vessel-wall.

Andral states that varicose veins occur in the pia and that they occasionally rupture. Capillary hemorrhage may follow the plugging of a large vein, and of the larger vessels any one or more may be involved, but it has been observed that hemorrhage tends to take place at particular places. In more than one-half of all cases the lenticulo-striate artery (Charcot's artery of cerebral hemorrhage) gives way, and damages the lenticular nucleus and internal capsule. Other regions in the order in which hemorrhage occurs are as follows: centrum ovale, cortex, pons, peduncle, cerebellum, optic thalamus, and the posterior and anterior parts of the hemispheres. Hemorrhage into the cerebrum occurs twenty times more often than hemorrhage into the cerebellum; it may take place into the brain-substance, into the ventricles, or into the meninges, the latter form having already been considered. Ventricular hemorrhage in a great number of cases is caused by a more or less extensive laceration of brain-matter, thus permitting the blood to escape into the ventricles. Not only the lateral ventricles, but the third and fourth also, may contain blood.

**Symptoms.**—Generally the patient is seized without any warning, but in other cases *headache*, *depression*, possibly *choreaform movements*, and more or less *paresthesia* precede an attack. The loss of consciousness is usually the first manifestation, though for a few moments before, motor weakness, with or without spasmodic movements, may exist. In very slight attacks consciousness may be preserved throughout. The cause of the *unconsciousness* is still an open question. Niemeyer regarded it as due to pressure acting either directly upon the convolutions or by limiting the blood-supply. This view is scarcely tenable, however, for unconsciousness occurs even when the hemorrhage is too small to exert pressure, and, moreover, the hemorrhage and loss of consciousness are usually simultaneous. The symptoms are in direct proportion to the extent and position of the hemorrhage. The patient falls, the face is usually congested, one side often expressionless, and the cheek flaps during respiration. Breathing is stertorous and, in grave cases, of the Cheyne-Stokes type; the pulse is generally feeble for a few moments, but soon becomes full and bounding in character. The pupils vary, and may either be contracted or dilated. There is frequently a relaxation of the sphincters, and on raising the limbs it will be found that those of one side offer absolutely no resistance. The *temperature* is slightly lowered at first, but after a few hours rises to, or just above, normal. In grave cases it will either remain low or will mount up to 106° F. (41.1° C.) or even higher. Such cases are usually fatal. *Conjugate deviation* of the head and eyes takes place in some cases, the deviation being toward the lesion and away from the paralyzed side; in pontine hemorrhage the opposite to this occurs. As a rule, the symptoms that we group under the term apoplexy—viz. loss of consciousness, motor power, and sensation, with or without relaxation of the sphincters—pass off in twelve to twenty-four hours. In fatal cases the coma deepens, but death rarely ensues under twelve hours. In hemorrhage into the medulla or ventricles it may be more rapid.

In from twenty-four to forty-eight hours after the onset *febrile reaction* sets in, with irritative symptoms due to the inflammatory changes occurring about the original lesion. There are fever, often delirium, twitchings or spasmodic movements of a more pronounced type, and sometimes rigidity in the affected limbs. *Trophic changes* in the form of vesicles, or even sloughing, may occur. Death may take place during this stage. Cases are generally fatal also in which a second "stroke" follows closely upon the first, indicating a fresh hemorrhage. After the reactionary period a stationary period follows; sooner or later control of the damaged members is then gradually, but not perfectly, regained. The degree of recovery is dependent upon the resumption of function of slightly damaged tissue or upon the compensatory activity of the other side of the brain. In well-marked cases the movements of the affected side are subsequently ataxic. In certain cases the structural damage has been too great, and permanent paralysis remains, with rigidity, wasting, and secondary contractures.

**Ingravescent Apoplexy.**—In certain cases the onset is slow, consciousness being lost gradually. Coma deepens and the case, as a rule, terminates fatally.

**Simple Apoplexy.**—The term "simple apoplexy" is almost obsolete.

**Serous Apoplexy.**—The cases present clinical evidences of apoplexy, but the only *postmortem* finding is an excess of serum, and this is in no way responsible for the apoplexy. These cases probably belong in the same category as those just mentioned, but occur in old persons whose brains have atrophied.

**Hemiplegia.**—When this is complete one side of the face and the arm and leg of one side, generally the same, are all involved. The facial palsy is not complete, the frontalis and orbicularis oculi escaping. The tongue when protruded deviates toward the paralyzed side. As a rule the arm is affected to a greater extent than the leg, and, indeed, in some cases the face and arm may alone be paralyzed. The trunk-muscles always escape, possibly owing, as Broadbent suggested, to the functional union of the spinal nuclei of *the two sides* that preside over them, and, since they habitually act together; he supposed that they might be stimulated from either hemisphere.

*Sensation* is, of course, absent during the period of unconsciousness. Subsequent sensory disturbances are not constant for all cases. Spots of anesthesia often exist for a brief period, but hemianesthesia is rare. Occasionally dissociation of sensation is present, tactile sensation being preserved, whilst muscular and thermal sensation is lost or diminished. A lesion in or about the posterior part of the posterior limb of the internal capsule is specially prone to give rise to disturbances of sensation.

The special senses may be temporarily perverted or their functions in abeyance, but rarely do permanent disturbances occur. Transient crossed hemianopsia usually occurs, even if the optic tract has not been directly involved. More or less mental deterioration may be permanent, however. The deep reflexes are increased on the paralyzed side, and the superficial reflexes are absent.

**Crossed Hemiplegia.**—When a lesion occurs in the lower part of the pons the fibers of the facial nerve that are involved have already decus-

sated; hence facial palsy occurs on the same side as the lesion. The fibers coming from the cortex are implicated before their decussation, so that paralysis of the limbs occurs on the side opposite to the lesion. Lesion of the crus may lead to oculo-motor palsy of the same side, and palsy of the face, arm, and leg of the opposite side.

**Course and Terminations.**—As previously intimated, the course depends on the position and extent of the lesion. In the most extensive cases death rarely takes place under several hours. Hemorrhage into the medulla may prove fatal more quickly. In the slightest cases, perfect recovery may take place in a few days or weeks. Generally, however, when little or no improvement occurs in two or three months, permanent changes result. The facial muscles soon recover, and next the leg, while at first the patient is able merely to move the toes. Daily improvement then follows until he can support his weight; dragging of the foot, however, is marked at first, and rarely disappears absolutely. In the mean time a less pronounced change for the better has been taking place in the arm. This member very rarely recovers, however, to the same extent as the leg, and secondary contractures develop in time, the arm and hand becoming flexed, whilst the leg is extended. The hand is usually bluish and cold, and swells if kept in a dependent position. More or less ataxia is constant, and rheumatoid pains are apt to occur during this stage. Other late manifestations that are only occasionally met with are athetosis, arthropathies, post-hemiplegic chorea, and tremors.

There is no wasting of the affected muscles as a rule; nor are there electric changes, except during the irritative period, when the response to stimulation is heightened. Occasionally marked atrophy occurs, and is due in some cases, as Charcot has shown, to changes in the cells of the anterior horns. In others no such change is found, and we are forced to regard the wasting as cerebral.

**Differential Diagnosis.**—Apoplexy is to be distinguished from other conditions causing unconsciousness, such as cardiac syncope, epilepsy, alcoholic or opium-poisoning, insolation, or uremia. If some previous history can be obtained, the difficulty of the diagnosis is lessened, though it may still be great. In *syncopal attacks* the pulse is very feeble and the face is pale, respiration being shallow and often suspended. The sphincters are hardly ever relaxed; the reflexes are usually preserved, and the skin is often moist. In *epilepsy* there is a history of previous attacks, or, failing to obtain this, one can usually learn that a convulsion has immediately preceded the coma. With *alcoholism* the case is more difficult. The odor of alcohol on the breath is of no value, as spirits may have been given by a bystander; moreover, hemorrhage is common in alcoholics (*vide* table of differential diagnosis, p. 965). In *opium-poisoning* the coma comes on gradually, and when not too profound the patient can be aroused when shaken or shouted at. The respirations, which are very slow and deep at other times, become somewhat quicker and shallower when he is aroused. In *insolation* the temperature suffices as a rule, though, as stated, high temperature may occur in apoplexy. The presence of albumin is not conclusive evidence of *uremic poisoning* unless the centrifuge and the microscope reveal the presence of casts or other indications of renal change; even then



the case may be one of apoplexy in a subject of nephritis. In the case of *diabetic coma* the presence of sugar in the urine serves to make the diagnosis. When we meet with a comatose case in which there is absolutely no resistance when the limbs of one side are raised, while those of the other still exhibit some tonic, particularly if the deep reflexes are exaggerated on the flaccid side, the probability is that it is an apoplectic attack. It is generally not possible to tell whether the condition is due to hemorrhage, embolism, or thrombosis, though the tabulated points of distinction (after Leube, *vide infra*) will afford material aid:

## EMBOLI.

Early adult life.

Previous development of atheroma, cardiac disease following acute rheumatism, sepsis, chronic valvular disease, fatty heart, general cardiac weakness, aneurysm.

Detection of emboli in other organs.

During the attack there is an absence of congestion of the face; the pulse is normal; in cardiac affections it is accelerated and irregular.

Temperature normal, but shortly after the attack it begins to rise, without making an unfavorable prognosis.

The attack, as a rule, is short, but if there is a protracted embolic infarction, the duration is long; but the circulation may adjust itself.

Remote effects are infrequent. Hemiplegia is right-sided, with aphasia.

## HEMORRHAGE.

Late adult life; in early life very rare.

Atheroma with cardiac hypertrophy.

History that the patient up to the time of attack was well; also the finding of casts in urine and other symptoms of chronic nephritis.

During the attack there are noted flushes (reddish) of the face, pulsating carotids, and slow pulse.

Temperature during the attack is subnormal, but just previous to death there is an *antemortem* rise.

The duration is, as a rule, longer. Coma of long duration (about two days) gives a very unfavorable prognosis.

Remote effects quite frequent; alteration in the urine—albuminuria, polyuria.

*Ophthalmoscopic Examination.*

At times the ophthalmoscope reveals either a recent or an old embolus in the arteria centralis retinæ.

The retinal arterics may show various stages of atheromatous degeneration; as a result there may be a hemorrhagic retinitis or there may be a thrombus of the central vein of the retinæ. In a few instances, in which the hemorrhage occurred in the ventricle, the pupils were contracted.

It is not an uncommon occurrence to have patients brought to a hospital dazed and smelling of liquor. These should always be carefully watched, for mistakes readily occur, and many such cases have been condemned to a prison-cell when they were really suffering from fracture of the skull.

**Prognosis.**—Sufficient has already been said on this point.

**Treatment.**—The patient should be kept as quiet as possible and in the recumbent position, with the head somewhat elevated. The clothing about the neck should be loosened to prevent constriction. An ice-bag may be put to the head and hot bricks or a hot-water bottle to the feet, while sinapisms may be placed on the back of the neck or on other parts of the body. If the pulse is strong, full, and incom-

pressible, and the face is congested—venesection is probably justifiable, particularly if the age and condition of the vessels support the idea that hemorrhage is taking place. It must not be done without consideration, however, since it would be useless in embolism and thrombosis. The bowels should be made to move freely; a cathartic may be exhibited by the mouth (croton oil, gr. j or ij), and at the same time an enema may be given. When consciousness returns the patient should be kept absolutely quiet for several days, and only liquid food permitted. Later an endeavor should be made to keep up the tone of the affected muscles by massage and electricity. It is questionable if the iodids or any other drugs have an influence over the subsequent changes.

### APHASIA.

**Definition.**—Impairment or total suppression of the power of speech, due to cerebral disease. This is a complex subject, and cannot be more than touched upon here; but the chief disturbances will be briefly mentioned, omitting any further description of the form due to disease of the bulb (*anarthria*), since it has already been dealt with in speaking of Bulbar Palsy (*vide* p. 1094).

The majority of cases of aphasia are met with in connection with hemiplegia. They are apt to be more marked in the beginning, owing to the general obtunding of the psychic processes that is induced by the shock. Speech is the expression of thought in words, and is the result of external stimulation in which the impulse awakens in the mind a recollection of similar impulses that have preceded it—*e. g.* the sight of a dog, sound of a bell, or certain odors (*vide* Fig. 72). Bastian, however, believes that it is not necessary to postulate the existence of a separate center for conceptions or ideas. He believes that a better knowledge of the functional activity of the auditory and visual word-centers obviates it, and gives the following three ways in which the perceptive center may be called into activity: (1) By voluntary recall of past impressions, as in an act of recollection; (2) by association—that is, by impulses communicated from another center during some act of perception or during some thought-process; and (3) by means of external impressions.

Two principal forms of aphasia exist: (1) *Motor*, and (2) *Sensory*. I shall consider these forms of aphasia separately, although before doing so it seems necessary to indicate by means of the accompanying schematic diagram (Fig. 73) the different paths on which the individual acts for the occurrence of speech are fulfilled. This will also serve to furnish an easy explanation of the symptomatology of the individual forms of aphasia and the means by which their discrimination (from each other) is possible.

**Motor Aphasia.**—As stated, we meet with aphasia most frequently in hemiplegia. It is also met with in certain cases of embolism and thrombosis and as a result of softening from any cause; also in certain cases of brain-tumor and in deaf-mutes. The latter cases are not strictly included in the definition given, for, while brain-injuries at birth may and do occasionally cause aphasia either by damaging the brain as a whole (imbecility) or in a more circumscribed fashion, I refer particularly to those cases in which the child is aphasic because he is deaf, for unless trained

through his visual or tactile sense he has no memory-picture of words from which to draw. A child may acquire the power of speech and retain it for several years, and then become deaf. He will then receive no more auditory impressions, and, unless trained as in the preceding group, will become aphasic. Certain cases of congenital speech-defect have been described by Hodden under the name "idioglossia." Here the children utter peculiar sounds, constant for the same words. As a rule, their intelligence is not up to the normal. In hemiplegia the lesion is usually in the motor, or emissive, center in the foot of the third frontal convolution (Broca's region). In right-handed people this is on the left

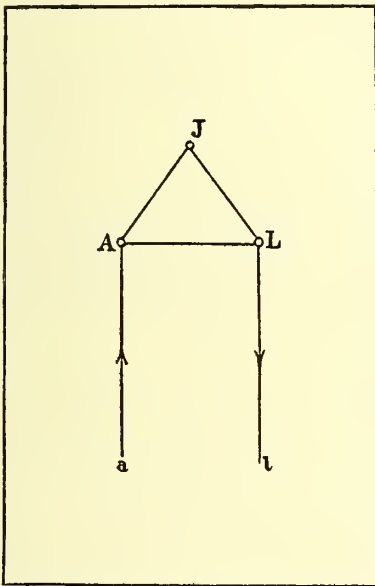


FIG. 76.—Diagram for the explanation of the process of speech (Leube): *A*, auditory perceptive center (center for the recognition of sounds); *a A*, acoustic tract (auditory tract); *L*, center of motor speech; *L l*, motor speech tract (path for the innervation of the muscles of phonation); *J*, center of ideation (higher concept center).

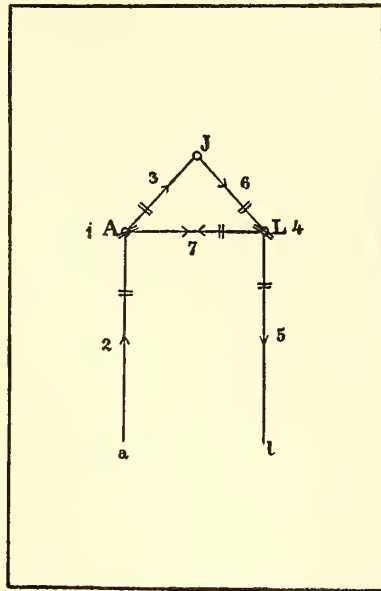


FIG. 77.—Diagram for the explanation of aphasia (Leube): *A*, *a A*, *L*, *L l*, *J*, the same as in Fig. 71; *A J L A*, circle by which speech is controlled; *a A L l*, path used for automatic speech; *J L l*, path used for voluntary speech; *a A J*, path used for the recognition of words; —, interruption of conduction; 1-3, forms of sensory aphasia (1, cortical; 2, subcortical; 3, transcortical); 4-6, forms of conduction-aphasia (1, cortical; 2, subcortical; 3, transcortical); 7, conduction-aphasia (amnesic aphasia).

side of the brain, and on the right side in left-handed people. Bastian believes Broca's region to be sensory and not motor (*vide* Sensory Aphasia, *infra*). A lesion in Broca's region causes the loss of voluntary speech, nor can the person repeat words after any one or read aloud; but he can understand when spoken to, and, unless his arm is paralyzed, can usually write from a copy. It is rare, however, for a lesion to involve so strictly limited a portion of the cortex. As a rule, adjacent parts or commissural fibers between neighboring motor centers are involved, resulting in "combined motor aphasia." In either case automatic speech is perfect, and oaths or other expletives may be uttered, songs sung, or poetry recited unless the oro-lingual centers are damaged.



Some observers claim that a special center for writing exists at the foot of the second left frontal convolution. Déjerine denies this. In some cases the patient comprehends when spoken to, and even speaks himself, but he misplaces words, and hence fails to convey his meaning. The lesion in such cases of paraphasia is believed to be in the commissural fibers uniting the frontal and temporal lobes, and is called "Wernicke's aphasia of conduction."

**Sensory Aphasia.**—This comprises various auditory and visual disturbances of speech.

(a) **Auditory Disturbances.**—The cortical center for auditory impressions is in the posterior part of the first and second temporal convolutions. That this region represents several auditory impressions—viz. that for words, music, and, in fact, sounds of any kind—we have abundant clinical proof. Cases are reported in which the patient recognizes all but spoken words. He can read and write, but when spoken to, hears the words just as one may hear a foreign language, but cannot understand. He cannot, therefore, write to dictation. This is "word-deafness." Prof. Edgren of Stockholm has reported cases in which the patient is tone-deaf. The sound is heard, but is not appreciated as music. While these various auditory impressions may occur singly, we most commonly meet with a condition in which they are all lost—"mind-deafness"—and in every case, except those possibly of the most trivial kind, more or less apraxia (loss of knowledge of the use of things) exists contemporaneously.

Bastian gives four centers for word-memory as follows:

(1) Auditory memory, the memory of the sound of words, situated in the posterior part of the first temporal convolution.

(2) Visual memory, the memory of the visual appearances (printed or written) of words, situated in the angular and supramarginal gyri.

(3) Kinesthetic memory, the memory of the different groups of sensory impressions, resulting from the mere movements of the vocal organs during the utterance of words (impressions from muscles, mucous membranes, and skin). This he places in Broca's region, and terms the glosso-kinesthetic center.

(4) The cheiro-kinesthetic center, not definitely located, he regards as the memory-center of the different groups of sensory impressions emanating from muscles, joints, and skin during the act of writing individual letters and words.

Bastian does not regard these four centers as sharply defined. He says on this point: "Although I am not a believer in the complete topographic distinctness of the several sensory centers in the cerebral hemisphere, I consider it clear that there must be certain sets of structurally related cells or fiber-mechanisms in the cortex whose activity is associated with one or another of the several kinds of sensory endowment." Such diffuse but functionally unified nervous networks may differ altogether from the common conception of a neatly-defined "center," and yet for the sake of brevity it is convenient to retain this word, and refer to such networks as so many "centers."

(b) **Visual Disturbances.**—The visual perceptive center of Monk is situated in the occipital lobe in the region of the cuneus and of the calcarine fissure. The visual memory-centers are in the angular and supra-

marginal convolutions. By the visual memory-center is meant that part in which are stored memories of things, faces, or places. Memory is no longer regarded as a definite and fixed unity, but rather as a number of memories, each being closely related cortically. Of this also we have abundant clinical proof. Kussmaul was the first to point out that blindness for words can exist as a separate entity, and for this he proposed the term "word-blindness." Letter-blindness can also exist alone. Charcot speaks of a patient who knew Latin, Greek, German, French, and Spanish, but lost the memory of some of the Greek and German letters only. In other cases the patient may recognize the letters, but cannot form them into words or sentences. Note-blindness, according to Professor Edgren, is a variety of amnesia in which the patient can no longer recognize musical notes, though he sees them as well as ever. In still other cases the visual memory for objects, faces, or places may be lost. The following case reported by Serieux is interesting in this connection: A woman aged sixty-two had a stroke, followed by paralysis, and after a second attack epileptic convulsions and hallucinations of hearing. She was found to have deafness and paraphasia, word-blindness, agraphia, and object-blindness (apraxia). She could not recognize the friends nor the objects that once were quite familiar to her. Some time later she died with pneumonia, and *postmortem* softening was found in the left first temporal (posterior part) and in the left supramarginal convolutions; also on the right side areas of softening were noted in the posterior parts of the first and second temporal convolutions and in the angular and supramarginal convolutions. This case illustrates the point of which I have already spoken—viz. the mixed character of the symptoms, due to simultaneous involvement of two or more centers. That this is the more common condition is not alone due to the anatomic propinquity of the various centers, but chiefly to the fact that they are all within the area supplied by the Sylvian artery and its branches. The simultaneous loss of these several visual memories results in the condition known as "mind-blindness." These sensory aphasias are sometimes spoken of as "amnesic aphasia."

Eskridge<sup>1</sup> has briefly summarized this subject as follows:

(1) If the lesion is in the foot of the left third frontal (Broca's) convolution in right-handed persons, and in left-handed persons in the right third frontal, the patient will be unable to speak voluntarily, to repeat words after another, read aloud, think in speech, and generally to write voluntarily or from dictation so as to be understood; but he understands the speech of another. He is usually able to use and to understand gesture-expression.

(2) A lesion in the oro-lingual center (lower portion of the central convolutions) will cause paresis or paralysis of the oro-lingual muscles, including the lower side of the face, and imperfect articulation; but the patient will be able, with a decided effort, to repeat words after another, to talk voluntarily, to think in speech, and to write, although the letters may be imperfectly formed. There is, as a rule, no omission or transposition of letters or words, unless the lesion is sufficiently extensive to affect the adjacent cortical centers or their commissural fibers. It is possible that the center is bilateral; that a lesion on either side may give

<sup>1</sup> *University Med. Magazine*, Jan., 1897.

somewhat similar symptoms; and that the loss of function in the affected muscles will not be completely abolished by a unilateral lesion.

(3) A lesion in the foot of the second frontal convolution on the left side in right-handed persons, or on the right side in left-handed persons (the probable orthographic center), will be attended by disturbances in writing—inability to write, due to inability to spell. In the only case of this kind that has been reported the patient formed all letters well, but omitted, transposed, and substituted letters to such an extent that his writing was unintelligible. He wrote well when words were spelled for him.

(4) A limited lesion in the posterior two-thirds of the first and second temporal convolutions is attended by word-deafness, and an inability to write at dictation (pure word-deafness). A more extensive lesion in the same region results in mind-deafness with paraphasia, and some disturbance in reading and writing will be added. The greater the extent of the cortical and subcortical areas involved the more marked the paraphasia and other symptoms of sensory aphasia.

(5) A lesion involving the angular gyrus and adjacent parts will cause word-blindness and inability to read, also defects in writing, copying, and speaking. In these cases paraphasia is often present. If the lesion affects the parts posterior to the angular gyrus, mind-blindness may be added.

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## INTRACRANIAL GROWTHS.

OWING to their close relationship, new growths, both of the brain and membranes, are here considered.

**Pathology.**—Rindfleisch has classified intra-cranial tumors according to the tissue from which they spring, thus:

1. Having their origin in the *membranes*, either extra-cerebral or intra-ventricular; these include tubercle, gumma, carcinoma, sarcoma, myxoma, lipoma, cholesteatoma, and psammoma; small fibroids have also been described. Enchondroma and osteoma may arise from the falx or from the bones of the skull.

2. From *blood-vessels*: to this group belong aneurysms, tubercles, and gummata.

3. Originating in the *neuroglial tissue*: glioma.

4. Originating in the *connective tissue*: sarcoma.

I will here consider these new growths in the order of frequency with which they are met.

1. **Tubercle** is most common in children and young adults, and is generally multiple (see Tuberculosis, p. 315).

2. **Sarcoma** is usually of the round- or spindle-celled variety; there may also be melanotic lympho- or fibro-sarcomata. Sarcomata are apt to diffuse themselves through the brain-substance quite rapidly.

3. **Glioma**.—Infiltrating tumors, generally single, and showing no definite line of demarcation from the surrounding brain-structure. They may be soft, even telangiectatic, or quite firm. They often run a very chronic course.

4. **Gummata** are generally small and often multiple. They spring



from the membranes or the adventitia of blood-vessels, or from connective-tissue septa. Frequently they are attached to the periosteum of the skull.

5. **Carcinoma** may be primary or secondary, usually the latter. Secondary growths are generally small and round, but in some cases they perforate the bones of the skull, producing a fungus hematoides.

6. **Fibromata** are not common. They either grow in the membranes or aid in the formation of a mixed tumor, as fibro-sarcoma. Other tumors met with less frequently are as follows: 7. **Osteoma**; 8. **Enchondroma**; 9. **Myxoma**; 10. **Lipoma**; 11. **Angioma**; 12. **Cholesteatoma**.

13. **Hydatids** are rare, and exceedingly so in America. They may develop in any part of the brain or its membranes, and are said to occur most frequently in children. 14. **Cysticerci** may also occur in the brain or its membranes.

15. **Brain-cysts** are probably most often due to absorption of areas of softening from any cause, but they also occur between the dura and skull, as has been described. The lack of cerebral substance, due either to imperfect development or to atrophy following vascular obstruction or injury at birth, has been termed *porencephalia* by Heschl.

**Étiology.**—Age and sex are the chief factors; tuberculosis is far more common in children than in adults, while gummata when found appear almost invariably in adults, as do malignant growths. As a whole, new growths are more common between the twentieth and fortieth years, and males are more often affected than females. Heredity and traumatism may be disregarded as causal factors.

**Symptoms.**—These are (1) General, and (2) Focal.

**General Symptoms.**—*Headache* varies in degree and character; it is not of any value as a localizing symptom, nor is tenderness on pressure.

*Vertigo* in a mild form is quite a common symptom. In cerebellar cases it is often very marked.

*Vomiting* occurs in most cases, and generally bears no relation to the time of taking food; this constitutes an important point in the diagnosis. The vomiting is apt to be exaggerated in cerebellar tumor.

*Optic neuritis* is present, according to Gowers, in four-fifths of all cases; in 82 per cent. according to Oppenheim, and in two-thirds according to Knapp. Gray believes this to be misleading in a measure. We are so imbued with the fact that optic neuritis is a symptom of brain-tumor that without it we are apt to overlook the possibility of tumor. He says it is unquestionably present as a rule in the later stages, but the practical question he asks is, "How often is optic neuritis in the earlier stages when the diagnosis is obscure?" Headache, vomiting, and optic neuritis are "classical symptoms" of brain-tumor, and when met with simultaneously are quite characteristic.

The *mental disturbance* is usually slight. Dulness and stupor are most reliable evidences of intra-cranial growth, and especially when occurring with any of the above symptoms. The patient may be emotional or hysterical. Pseudo-apoplexy may occur as the result either of the growth or of hemorrhage taking place about it.

*Convulsions* are focal (Jacksonian), or general convulsions may occur.

*Constitutional and other symptoms* may include progressive weakness, loss of appetite and of flesh, pupillary changes, and changes in the pulse,

respiration, etc., and possibly slight fever. In certain tumors in the basal ganglia hyperpyrexia is met with. High fever is often significant of meningeal inflammation, as in syphilitic cases.

The focal symptoms are of two kinds: first, those due to direct local action (irritation or compression), and second, those due to changes occurring about the growth—indirect irritation, hemorrhage or softening, or merely congestion; thus can intermission or remission in symptoms be explained. The chief regional symptoms are as follows in—

(a) Tumors in the *prefrontal region*. Headache, not limited to the frontal region, with more or less mental impairment and drowsiness (though this is not constant by any means); and perhaps a disturbance of the sense of smell. No motor or sensory symptoms are present as a rule. The tumor may, however, grow backward, and either encroach on the motor region or cause motor symptoms indirectly. Downward growth would result in aphasia.

(b) Tumors in the *motor region*. The early symptoms are irritative and, later, paralytic. The former give rise to *spasm*, which is often very localized at first, possibly in a few muscles (Jacksonian epilepsy). More or less *sensory disturbance*—*e. g.* tingling or anesthesia—is generally present also, thus supporting the view that the motor cortex is also sensory. The point of origin and direction of spread of the spasm are valuable localizing symptoms. Sooner or later destruction of the area causes *paralysis*. We may have spasm in one limb and monoplegia of the other on the same side. Involvement of the left third frontal region causes aphasia.

(c) Tumors in the *parietal lobes*. There may be no symptoms. When the posterior part is involved (angular or supramarginal gyri) we may meet with word-blindness or mind-blindness.

(d) Tumors in the *temporal lobes* may be latent, or there may be disturbances of taste and smell. If the posterior part of the first convolution is involved, we have word-deafness or other psychical disturbance of hearing.

(e) Tumors in the *occipital lobes*. A unilateral tumor produces *hemianopia*, while a bilateral lesion may cause blindness. In certain cases, too, mind-blindness results, or “soul-blindness,” as it was at one time called.

(f) Tumors of the *corpus callosum* are often latent; they may, however, cause unilateral or bilateral motor symptoms. Often some mental aberration is noted.

(g) Tumors of the *corpora quadrigemina* and crura may be considered together, since in the former case no distinctive symptoms occur when the tumor is small; when large the crura will be implicated. In such cases there will be oculo-motor paralysis on the same side as the tumor, and hemiplegia on the other. Sensory disturbances may occur also.

(h) Tumors involving the *base*, when growing in the anterior fossa, give rise to exophthalmos, disturbances of smell and vision, and possibly to mental impairment. When in the middle fossa the symptoms are chiefly those of involvement of the third and fifth nerve, consisting of ptosis and other oculo-motor symptoms and facial neuralgia. When the tumor involves the pituitary gland, amblyopia or amaurosis and frontal headache occur. Tumors have been found in this region in cases of

acromegaly (*q. v.*). When in the posterior fossa, facial neuralgia, neuro-paralytic ophthalmia, or seventh or eighth nerve involvement and crossed hemiplegia are met with.

(*i*) Tumors in or about the *basal ganglia*, if quite small, cannot be diagnosed. When of larger size they cause hemiplegia and sensory disturbances by pressure upon the internal capsule, and hemianopia by pressure on the optic radiation. They may also cause obstruction and consequent distention of the ventricles (internal hydrocephalus).

(*j*) Tumors in the *cerebellum* may be latent. They give rise to most marked symptoms when the growth is in the middle lobe. In such cases headache, vomiting, vertigo, and ataxy are pronounced. The gait is of a reeling or staggering character, and there is a tendency to fall in some particular direction—forward, backward, or to one side. In addition there may occur optic neuritis, disturbances of vision, pain and stiffness in the cervical region, and possibly deafness.

(*k*) Tumors in the *pons* and *medulla* give rise to cranial nerve-lesions, with or without hemiplegia; also to sensory disturbances, and when extensive they may by pressure cause cerebellar symptoms. Tumors in the medulla may cause cardiac or respiratory failure.

It is often important from a surgical point of view to ascertain whether the growth is cortical or subcortical. No absolute means of doing this exists, though Seguin has formulated a fair working rule—viz. when localized clonic spasm occurs, followed in some cases by epilepsy (this primary spasm Seguin has termed the “signal symptom”), and later by paralysis, with local pain and tenderness and increased local cerebral temperature, a cortical lesion is most likely. On the other hand, when the spasm is tonic, particularly if widespread—involving one side, for instance—and when the local pain and tenderness are slight or absent, and the cranial temperature normal, the lesion is more apt to be subcortical.

**Course.**—Many cases run a very chronic course. Others may have existed months or years without symptoms, and then develop suddenly, owing to hemorrhage, thrombosis, or acute softening about the tumor. Either improvement may take place or the case may speedily progress to a fatal termination.

**Diagnosis.**—The general symptoms are usually sufficient to warrant a diagnosis. The gradual onset and progressive character without fever, in the apparent absence of any etiologic factor, are, as a rule, enough to indicate that a tumor is present, while its location can only be determined by the focal symptoms.

The **prognosis** is always grave. Syphilitic growths are almost the only kind in which some gleam of hope may be extended, and this only very rarely; tubercle may recover by the growth becoming encapsulated and calcified. Nothing can be said as to the possible duration of life. Several years may elapse between the appearance of the symptoms and their fatal termination, or death may occur suddenly.

**Treatment.**—In any case recourse should be had to mercury and the iodids, and this treatment should be pushed, since it will certainly benefit syphilitic cases, and it is believed to be of some value even in the non-syphilitic. Other symptoms should be met as they arise. The question of operation must be considered where medical measures have proved of no avail. Macewen and Horsley in Great Britain and Keen



in this country have pointed out its justifiability, and, while it is admittedly *le dernier ressort*, the following table of Dr. R. B. Newton, taken from Gray, shows the comparatively large percentage of recoveries:

Total number of operations for tumor to Sept. 15, 1892 . . . . .	110
Times failed to remove the growth when found . . . . .	5
Times tumor not found at point of operation . . . . .	15
Times trephined or operated for relief of pressure . . . . .	7
Recovered after removal of tumor . . . . .	63
Number of deaths . . . . .	42
Percentage of mortality . . . . .	38 $\frac{2}{11}$

## CHRONIC HYDROCEPHALUS.

THIS affection is divided into external and internal hydrocephalus.

### EXTERNAL HYDROCEPHALUS.

**Pathology.**—When the skull is opened the bone is usually found to be thin; the dura is normal; the arachnoid is lifted from the surface of the cortex by a considerable accumulation of clear fluid of low specific gravity; the convolutions may be somewhat flattened, and the cortex slightly thinned. Upon microscopic examination no changes are found in the brain-substance. Sometimes the effusion is general; sometimes it is sacculated. In a case of epilepsy with very marked neuropathic heredity, upon which I recently performed an autopsy, two large subarachnoid cysts were found beneath the temporal lobes, and smaller cysts in other parts of the brain, flattening or separating the convolutions.

**Etiology.**—External hydrocephalus may depend upon a congenital smallness of the brain or upon a congenital enlargement of the skull. The space between the brain and the bone is filled by an excess of subarachnoid fluid (*vacuum dropsy*), or there may be a wasting of the brain, such as occurs in old age or in chronic cachectic conditions.

The **symptoms** depend upon the form. In cases in which there is hypoplasia of the brain or in which the brain has wasted, no pressure-symptoms are present. All the manifestations are purely psychic in nature, and similar to those of *microcephaly* or *senile dementia*. In cases, however, in which the cranium-cavity is abnormally large, it is probable that the real cause resides in a congenital excess of subarachnoid fluid.

The **prognosis** is gloomy; nevertheless, it is possible that the disease may undergo spontaneous cure as a result of rupture into the nasal fossa.

The **treatment** is the same as for the internal variety (*vide infra*).

### INTERNAL HYDROCEPHALUS.

This is a condition in which one or more of the ventricular cavities of the brain are distended by the cerebro-spinal fluid. In the *congenital form* and in that occurring in early childhood, this is associated with more or less enlargement of the skull. In the later *acquired forms* the cranium does not yield so readily, and the enlargement does not exist or is slight.

The **pathology** of the condition varies with its nature. In the congenital forms, upon opening the head the skull is found to be thin. The

fontanels and sutures are either still open and connected only by a membrane, or closed by Wormian bones. The dura may be thickened, but usually is normal; the substance of the brain is slightly softened—although this is not invariably the case—and very much thinned. This thinning is, as a rule, particularly noticeable in the corpus callosum and commissures, which may, indeed, either be torn apart or completely atrophied. The enlargement ordinarily affects the two lateral ventricles, the third ventricle, and the aqueduct as far as its entrance into the fourth ventricle, which is commonly less involved than the other cavities. The ependyma is sometimes smooth, but more often shows small projections, which, according to Virchow, are composed of brain-substance, but in some cases are due to proliferation of the glia tissue beneath the ependyma. The enlargement may not be uniform. If due to obstruction of the foramen of Monro, one or both lateral ventricles are usually enlarged, whilst the third ventricle either remains of normal size or is diminished. If due to enlargement of the pineal gland, the aqueduct does not show the funnel-shaped distention. Another cause upon which considerable weight has been laid is the closure of the transverse fissure between the cerebellum and medulla. The quantity of fluid may be enormous, as much as 4 or 5 liters (5 or 6 quarts) having been recorded. The thinning of the brain-substance is also remarkable when one considers that a cerebrum 5 mm. ( $\frac{1}{6}$  in.) in thickness is apparently able to perform a large proportion of its ordinary psychic functions. The atrophy seems to affect particularly the white substance, especially the myelin-sheaths.

In cases of the *acquired form*, unless they occur early in life, the enlargement of the skull is not very noticeable; the substance of the brain shows considerable softening; the ventricles are moderately enlarged, and, particularly in the chronic forms due to tuberculosis, are considerably roughened. The most pronounced cases are those that occur when there is a tumor in the occipital fossa which compresses the veins of Galen. In these cases the accumulation of liquid is slower, the brain yields more gradually to pressure, and the dilatation is more pronounced. Ordinarily, there is considerable flattening of the convolutions. In a few of these cases inflammatory changes in the ependyma have led to partial obliteration of the ventricles, particularly in the anterior horns or the lateral ventricles. Occasionally also bands of organized lymph may cross the ventricles in various directions; the liquid is of higher specific gravity and contains more albumin than in the non-inflammatory varieties.

The **etiology** of the *congenital form* is unknown, though the fact that it frequently occurs in several children of the same family has led to the supposition that it is dependent upon some hereditary influence. In some cases it has been referred to emotional disturbances suffered by the mother during pregnancy, and in still other cases an anatomic foundation has been discovered, such as enlargement of the pineal gland. It is generally supposed that the immediate cause is chronic ependymitis.

The acquired form is usually secondary to inflammatory conditions (particularly meningitis) or to brain-tumor. Some cases, however, occur in childhood, that are apparently not due to either of these causes.

**Symptoms.**—The most characteristic appearance in congenital hydrocephalus is the *globular enlargement of the head*. Upon palpation the fontanels are found to be still patulous and usually bulging, and the

sutures are open. The head is usually so heavy that it cannot be held upright, but falls backward or to one side. The face appears proportionately very small. *Motility* is usually disturbed, the legs are spastic, and the child either does not learn to walk at all or only long after the usual time. There are sometimes choreic movements of the upper extremities. The *eyes* frequently show nystagmus and conjugate deviation, and often there is either choked disk or atrophy of the optic nerve. Fischer has described a *systolic murmur* that can be heard if the stethoscope is placed over the anterior fontanel. Its cause is unknown. *Convulsive attacks* are common; they are epileptic in type, and, as a rule, ultimately cause death. *Intelligence* is usually considerably impaired, and sometimes the children are idiots; more often they merely show retardation of intellectual development. Occasionally—and this even in the most pronounced cases—the intelligence is well preserved. Henoch records the case of a boy three years of age whose head was 75 cm. (29.6 in.) in circumference, and who could speak both French and German. Ordinarily, the children are quiet and apathetic, but they may be querulous. *Nutrition* is commonly seriously disturbed, the children sometimes exhibiting pronounced cachexia. They may, however, be well nourished and, to a certain degree, vigorous. The symptoms of the chronic form in adults are those of brain-tumor without focal symptoms.

The **diagnosis** is ordinarily very easy. Careless observation may lead to confusion with *rachitis* but the square shape of the head and the presence of other rachitic deformities in the skeleton should lead to a prompt recognition of the true nature of the case.

The **prognosis** is extremely unfavorable, the majority of the children dying about the fifth year. A few cases, however, may live until they reach young adult life, and still fewer apparently recover entirely.

**Treatment** is of course difficult. Potassium iodid and mercury have been employed without much beneficial effect. Cod-liver oil may be given to stimulate nutrition, and purgatives occasionally relieve pressure-symptoms temporarily. Among the mechanical procedures constant pressure upon the head seems the most valuable. This can be obtained by means of strips of adhesive plaster or by the application of an elastic band. Tapping the ventricles is occasionally followed by temporary improvement, but is always dangerous. More satisfactory is probably the lumbar puncture recommended by Quincke. If convulsions develop, they should be combated by bromids and purgatives.

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## SCLEROSIS OF THE BRAIN.

THIS may be diffuse or focal. The diffuse form is usually a congenital condition, and leads to a hardening of the whole brain, associated with more or less malformation. If it is secondary to some vascular disturbance in early life, it may be restricted to one or more lobes on one side. The focal form occurs in insular sclerosis, and as a congenital lesion. The latter may be associated with overgrowth or contraction of the neuroglial tissue, and is spoken of as hypertrophic or atrophic gliosis. It is usually associated with epilepsy and idiocy. Neuroglial hyper-



plasia is also met with in areas in which the nervous tissue has been destroyed, no matter what the cause. It occurs, therefore, in both primary and secondary degeneration. The sclerotic areas vary in size from very small and even microscopic plaques to large and widely-diffused ones. These sclerosis have been considered under the respective diseases to which they give rise. Focal sclerosis, due to scar-formation and characterized by an overgrowth of connective tissue, occurs after injury or hemorrhage.

## GENERAL PARALYSIS OF THE INSANE.

(*General Paresis; Dementia Paralytica.*)

**Definition.**—A chronic disease involving both the cerebrum and the meninges, and characterized by a gradual loss of power, tremors, and progressive mental decay.

**Pathology.**—The intima and adventitia of the blood-vessels undergo proliferative changes, and the perivascular spaces are dilated and contain an excessive quantity of fluid, also cellular elements. Obliterative endarteritis occurs also. Atrophy and degeneration of the cerebrum are met with, chiefly involving the cortex, particularly that of the frontal or parietal regions and the anterior basal region. The ventricles are dilated and the ependyma is granular. The membranes are thickened and opaque, and adherent to the surface of the convolutions, so that the cortex is torn upon their removal. Hemorrhage may take place into the subdural spaces, and may vary in amount from a mere stain to the formation of a pseudo-membrane.

Secondary sclerotic and descending degenerative changes may be found in the cord in some cases.

**Etiology.**—As in locomotor ataxia, a history of syphilitic infection is obtained in a large majority of all cases. The condition occurs more frequently in men than in women, and usually between the thirtieth and fiftieth years. Business or domestic troubles, and, in fact, great anxiety of mind from any cause, also venereal or alcoholic excesses, serve more or less directly to induce the disease. Trauma and heredity play but a minor part.

**Symptoms.**—The **prodromal stage** may last for months or even years. The symptoms are both mental and physical, either of which may appear first and exist alone for some time, or they may be contemporaneous in point of onset. As a rule, some *alteration of the character* and demeanor of the patient is the first evidence of the trouble. The patient suffers from insomnia and is generally restless, as well as incapable of sustained effort. He will be forgetful and perhaps careless where he was formerly careful and attentive. The sexual passions are aroused, and the patient may suffer from a sort of erotomania, sometimes taking the form of perversion. The *ego* will figure prominently in his sayings and doings. Among the physical signs are frequent twitchings and *tremors* of the facial muscles, particularly of those about the mouth and the tongue. Tremors of the hand and arm seriously interfere with writing; tremor of the lips and tongue renders the *speech* thick, blurred, and hesitating, and syllables are omitted from words, or even whole words lost from

sentences; and the *pupils* are frequently unequal and fail to react to light. These symptoms extend over a variable period, with one or more remissions as a rule, and sometimes with a complete intermission and an apparent cure. Sooner or later, however, the next stage develops.

**Stage of Mania or Melancholia.**—The symptoms of this stage are superadded to those of the first, which by this time have grown gradually more pronounced; *loss of power* usually is already a prominent feature. Acute outbreaks of *mania* are most commonly met with, and are characterized by a most remarkable prodigality of thought and speech. The patient believes himself to be possessed of enormous wealth or of great rank and power. He is boisterous, sleepless, and constantly and actively engaged in pursuing his extravagant ideas. In other cases this delusion of grandeur (*expansive delirium*) is absent and the patient is melancholic. This is especially apt to be the case if his physical condition is lowered by some intercurrent disease. Remissions of all these symptoms are not rarely met with. Whether the prevailing type be that of mania or melancholia, *paroxysms of terror* may occur from time to time, or mania and depression may alternate. *Epileptiform* or *apoplectiform attacks* occur, followed by paralysis. In the large majority of cases the mental decay is progressive, until finally complete fatuity is reached; the patient then becomes bedridden, bladder and rectal symptoms develop, and possibly bed-sores. Death results from exhaustion or from some intercurrent disease.

**Diagnosis.**—This is always difficult, and it is often impossible to diagnose the disease in the earliest stages, particularly when the mental phenomena alone exist. The slight change of character and the occasional outbursts of temper or unrestrained jollity may be regarded as mere moods more or less directly dependent upon the daily routine. When to these symptoms are added the tremor, the defects of speech, the inequality of the pupils, and paresis, the clinical picture gradually assumes definite shape, and oftentimes, long before expansive delirium or melancholia develops, a positive diagnosis is made.

**Differential Diagnosis.**—The diseases with which it is most likely to be confounded are—(1) *Disseminated sclerosis*, (2) *Paralysis agitans*, and (3) *Cerebral syphilis*.

(1) In *disseminated sclerosis* the mental symptoms are even less obtrusive in the earlier stages, the first evidence of the disease being paresis in the lower extremities. The tremor too is volitional, the speech is scanning, and nystagmus is present. The reflexes are exaggerated, and there may be spasticity. Mental phenomena generally develop later.

(2) In *paralysis agitans* there are frequently no mental changes, and in any case they consist of nothing more than dulness. The characteristic attitude and gait; the tremor when at rest, which sometimes ceases on movement; the speech, which is hesitating at first, then hurried; the high-pitched voice; the absence of pupillary changes,—all mark paralysis agitans. Remissions are uncommon.

(3) *Cerebral syphilis* may also simulate paretic dementia. The differential diagnosis is comparatively easy in most cases, however, and particularly if the early history can be obtained. In cerebral syphilis the tremor may or may not be present, but no speech-defect occurs; epileptiform attacks are common (usually *petit mal* or Jacksonian), and attacks of head-

ache are frequent and usually severe. The condition often passes into dementia, and then closely resembles general paresis.

The **prognosis** is gloomy, and recovery very seldom occurs. The tendency is toward a fatal termination in from two or three to fifteen years.

**Treatment.**—Drugs are of no value in a curative sense, except in those cases that are due to syphilis, when the iodids must be pushed. Bromids, morphin, chloral, or, still better, sulfonal or trional, may be used in combating the insomnia and attacks of delirium. These cases cannot be properly cared for at home; indeed, their removal to an asylum is generally imperative. The tendency to bed-sores must not be forgotten, and continuous rest in bed must therefore be postponed as long as possible.

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## CEREBRAL PALSIES OF CHILDHOOD.

**Definition.**—This includes hemiplegia, the birth-palsies of Gowers (diplegia), and paraplegia, each being characterized by a particular palsy, and in certain cases with rigidity, and all showing more or less mental defect.

1. **Hemiplegia.**—*Pathology.*—Practically nothing is known of the early changes that take place in most of these cases. They are essentially chronic, and do not reach the autopsy-table until late in the disease. A minority of cases, due to some vascular disturbance—embolism, thrombosis, or hemorrhage—may be clear enough. The others have been ascribed to some one of the following lesions: (1) Polio-encephalitis of Strümpell, who advances the view that there is an acute inflammation of the motor cortex similar to that of the motor cells of the anterior horns. (2) Congenital encephalitis of Virchow, believed by him to be an interstitial inflammation, with the formation of yellowish bodies in the white substance, due to the deposition of fatty granules. (3) Meningo-encephalitis, with opacity, thickening, and adherence of the membranes. The later changes are as follows:

(a) *Atrophy or Hypertrophy and Sclerosis.*—The sclerotic areas vary from small plaques to extensive regions, involving even the entire cortex of one hemisphere. They are hard and firm, and in the hypertrophic variety stand out beyond the normal tissue-line. The blood-vessel walls generally show proliferative changes, and the membranes are generally thickened and adherent, though in some cases they show very little if any change.

(b) *Porencephalia.*—This condition, first described by Heschl in 1859, is etiologically very obscure. Among the causes said to give rise to it are arrest of development, vascular disturbances, encephalitis, and hydrocephalus. It consists of a loss of cerebral substance, cysts of various kinds extending into the brain-substance and reaching even to the ventricles. An entire hemisphere may sometimes be wanting, and the cranium is sometimes distorted.

**Etiology.**—Enough has been said to indicate the uncertainty as to the cause of this condition. Many cases are congenital, and the large



majority develop within the first three years of life. There may be a history of dystocia, with or without the use of forceps. Cerebral traumatism and the infectious diseases are cited as having a more or less direct etiologic relationship.

**Symptoms.**—In many cases the symptoms are similar to those met with in hemiplegia of adults. Without premonitory symptoms a *loss of consciousness* develops suddenly; in other cases local or general *convulsions* precede the unconscious period; while in still another group consciousness is never lost. *Fever* is always present, but does not go above 102° F. (38.8° C.). The *hemiplegia* may be of gradual onset, though usually it is found to be quite pronounced when consciousness returns. The face is generally not involved, and even when it is implicated the upper parts (eyelids and forehead) escape. The right side is affected most frequently, and the upper extremity to a greater extent than the lower. Just as we see in adults, so it is in children. The paralysis may clear up in a few days, leaving little or no trace behind, the rule being for the leg to recover first. The arm may or may not recover. Where permanent damage is done the muscles waste, though usually but little. Neither sensory nor electric changes occur. The reflexes are increased and spasticity may develop (*hemiplegia spastica cerebialis* of Heine).

These children are, as a rule, mentally deficient, varying from what may be regarded merely as stupidity to idiocy or imbecility. Speech may not be acquired until late or not at all. It is probable, too, that the cases of “idioglossia” and congenital speech-defect, first described by Hadden, belong to this category.

Various forms of post-hemiplegic movements are quite common. These may be tremors, choreiform movements, or athetosis, and about half of these children develop epilepsy. Occasionally the latter is Jacksonian in type; usually, however, it is *petit mal* or *grand mal*.

**2. Diplegia, Birth-palsies, and Paraplegia.**—These conditions are described together, since they have the same pathologic substratum. They are characterized by double hemiplegia or paraplegia, increased reflexes, often by spasticity, preservation of sensation, and a lack of mental development. While hemiplegia may or may not be congenital, these cases of congenital spastic paraplegia (or “Little’s disease,” as it is sometimes called after an orthopedic surgeon, one of the earliest writers upon it) always date from birth, though the condition may not be recognized until months later. Probably most cases result from meningeal hemorrhage, due to the use of forceps or other injury at birth, as pointed out by Sarah J. McNutt, or perhaps to a previous meningo-encephalitis. At all events, whatever the earlier changes be, the later ones are those of atrophy or porencephalia. Van Gehuchten has published some interesting papers recently on this type of infantile palsy. His conclusions are that it is met with most frequently in children born prematurely, no matter whether the labor be a difficult one or not. He calls attention to the fact that at this early stage the pyramidal tracts are non-medullated, and therefore unable to functionate normally. The pons-cerebellar pathway is the first to myelinate, the last being the fibers of the direct motor pathway, which become medullated from above downward; hence those to the lumbar and sacral region are last of all, and may never become covered. This is in keeping with our clinical findings. The palsy may dis-

appear from the face, then from the upper extremities, and, finally, or perhaps not at all, from the lower extremities.

**Symptoms.**—In some cases a history of convulsions with febrile attacks is obtained; in others nothing abnormal is observed until the child commences to walk and to try to use its arms in a definite manner. The limbs will be found more or less rigid, and in a few cases the face-, head-, and neck-muscles will be affected. The *reflexes* will be increased, though sensation is generally unchanged. The *mental condition* is almost always very poor. Various grades of spasmodic incoördination are met with; also choreiform movements and athetosis.

The **diagnosis** is not difficult if we can obtain a definite history; otherwise it may be. The absence of electric changes will serve to differentiate it from anterior polio-myelitis.

**Prognosis.**—The extent and degree of paralysis and the character of the mental change are important aids in forming a prognosis. Taken as a whole, the outlook is not particularly bright in any case.

**Treatment.**—Apart from the treatment of the early convulsions, which must be promptly met by hot mustard baths, enemata, purgatives, and the bromids, little can be done. Massage and faradization may be tried. In view of the results of operative measures these are unjustifiable, nor could we expect to gain much, if anything, owing to the nature of these cases. Careful and systematic training is of great importance, for, while some remain imbeciles, others, though very slow to learn, ultimately reward the patience of the teacher.

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## ACUTE DELIRIUM.

(*Acute Delirious Mania; Typho-mania; Acute Periencephalitis; Bell's Mania.*)

**Definition.**—An acute maniacal delirium associated with hallucinations, with a febrile course, of limited duration, and of grave prognosis.

**Pathology.**—According to older writers, lesions were not found in the brain, but more accurate methods of microscopic research have shown that minute pericapillary hemorrhages are almost invariably present. In addition to these there are usually degenerative changes in the ganglion-cells. Often injection of the pia and minute hemorrhages into the gray matter may be observed with the naked eye. Cramer has recently reported a case in which the pericapillary spaces of the brain were filled with mononuclear leukocytes, surrounding which were recent hemorrhages; he also noted the fact that the ganglion-cells, instead of exhibiting normally-formed chromophilic bodies, were filled apparently with fine dust.

**Etiology.**—The disease occurs in either sex with about equal frequency. Predisposing conditions are neuropathic heredity, nervous disposition, the presence of other nervous diseases, particularly neurasthenia and epilepsy, alcoholic or sexual excesses, and severe prolonged anxiety. It has been supposed (Hertz) that abnormal narrowness of the jugular canal, which has been noted in several cases, bears some etiologic relation to it. It frequently occurs apparently as the immediate result of menstruation, parturition, injuries to the head, sunstroke, acute infectious diseases, particularly pneumonia and typhoid fever, and it may

develop in the course of chronic mental diseases. Occasionally, however, it appears to arise without any definite cause.

**Symptoms.**—The disease usually commences with certain indefinite prodromes. These consist of *restlessness*, associated either with melancholia, preoccupation, or anxiety. The *intelligence* becomes distinctly decreased; the patient loses appetite, is constipated, and commences to emaciate. During sleep unpleasant dreams or nightmares almost invariably occur. Sometimes there is a sense of impending mental disorder. This period gradually changes to one of defiance, which perhaps, even in the prodromal stage, may lead to violence and injury to those in the neighborhood. The prodromal stage rapidly passes to *acute delirium*, in which two steps may be recognized—excitation and collapse. The excited stage commences suddenly; there is great confusion; the patient ejaculates disconnected sentences or words or even syllables. There is great anxiety, and even fear, and the patients exhibit intense excitement, suffering very often with delusions of persecution by their environment, and nearly always having hallucinations, either of sight or sound. Often their minds are occupied by some subject that had previously caused them great anxiety—either disgrace, business, or other misfortune. The mania is often dangerous; indeed, it is likely that the disease known as “running amuck” in the Malay Peninsula is simply one of the forms of acute delirium. The patient soon becomes extremely restless, throws himself from one side of the bed to the other, and makes efforts to rise and escape from the room. The tongue is dry, the pulse rapid and weak. Petechiæ may appear upon the skin, and there is nearly always more or less fever, not rarely rising to  $105^{\circ}$  ( $40.5^{\circ}$  C.) or even more. Rapid emaciation supervenes. There are all the objective symptoms of irritation of the brain—myosis and increased reflexes, and often hyperesthesia, although the patients pay little attention to any injury they may inflict upon themselves. Sometimes there seem to be curious imperative movements; at others, imperative ideas. In a case that I observed the patient rhymed, very imperfectly it is true, each two successive sentences. This stage of excitation soon passes into one of *stupor* and *collapse*; fever may become even higher, and the pulse still more rapid and weaker. The patient lies in a condition of muttering delirium, with carphologia. All the symptoms are those of profound exhaustion: the eyes are hollow, the lips and teeth covered with sordes, and the emaciation extreme. The skin becomes dryer, and finally cyanotic, the pupils dilate, and there may be marked anesthesia. Death ordinarily occurs at the end of two or three days after the commencement of this condition. Occasionally the course of the disease is interrupted by intervals in which the patients exhibit more or less lucidity. Certain varieties have also been described. Thus in addition to the maniacal form authors speak of the *melancholic* and *paralytic* forms. In the former of these the patients exhibit, in place of excitement, profound depression, with fear of poisoning and positive refusal of all food; slight elevation of temperature, or, indeed, a sub-normal temperature, and very rapid emaciation. It is most apt to occur in patients previously debilitated. In the paralytic form there is vasomotor paralysis with cyanosis, depression, and often stupor. From these the patient passes into an algid state, in which death occurs.

The **differential diagnosis** is frequently difficult. In many infec-



tious diseases, particularly *pneumonia* and *typhoid*, hallucinatory delirium may develop. This, of course, must be suspected in these diseases, and it is advisable, if possible, to examine the blood in all cases of acute delirium by Widal's method. In *acute mania* fever is rare, emaciation is not so rapid, and the mental symptoms are more purely psychical. In *general paralysis*, toward the end maniacal attacks may develop, but the history of the previous existence of the disease, the presence of the Argyll-Robinson pupil, and the absence of fever lead one to suspect the true diagnosis. Finally, in *delirium tremens* the fine tremor of the hands and tongue, and, if possible to obtain it, a history of recent debauch, should clear up the diagnosis. The course of the disease is variable; it may vary from three or four days to as many weeks. Those cases are most rapid in which excitation is most profound.

The **prognosis** is most unfavorable, and is more so for men (according to Krafft-Ebing) than for women. Those cases that were previously debilitated, either as a result of chronic alcoholism, or chronic exhaustive diseases, or childbirth, are the most serious. Those that develop suddenly, and from the beginning are very severe, are also nearly always fatal; if there are no lucid intervals, or if those that occur are short and imperfect, the prognosis is graver; and the same is true of those who suffer from obstinate insomnia.

The **treatment** is of course unsatisfactory. In spite of progressive exhaustion, bloodletting is recommended, and calomel should also be administered in the earlier stages of the disease. At the same time the temperature should be combated by cool baths, and an ice-bag should be applied to the head. Sleep should be obtained by the use of chloral, bromids, and the more modern hypnotics, which are to be preferred to morphin. Hyoscin seems to be particularly indicated. In the later stages of the disease stimulants should be administered freely. Excellent results have been obtained (Solivetti) by the hypodermic administration of Bonjean's ergotin. Nutrition must be maintained by forced feeding with milk, eggs, broths, etc.

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#### IV. DISEASES OF UNKNOWN PATHOLOGY.

##### EPILEPSY.

**Definition.**—A condition characterized by attacks of unconsciousness, with or without convulsions. We are scarcely justified in speaking of epilepsy as a disease. It seems, in reality, to be a symptom, though in many cases (the so-called idiopathic cases) we know not the underlying cause. The type of cases in which the unconscious period is very brief (momentary), with no convulsion following or at most but a slight rigidity, is termed *petit mal*. The more pronounced type, with prolonged unconsciousness and severe general convulsions, constitutes *grand mal*. That form first described by Hughlings Jackson in which the convulsion is localized, and in which unconsciousness may or may not occur, is called *Jacksonian, focal, or cortical epilepsy*.

**Pathology.**—Gray regards epilepsy as a symptom, and if this theory

be correct, the inevitable question must be, "Of what?" In certain cases this can be answered (in the organic cases), since the lesion is demonstrable; but in others (functional or idiopathic) there is no demonstrable lesion. Among the causes of the former are brain-tumors, meningitis, traumatism inflicted either at birth or subsequently, atrophy and sclerosis, vascular disturbances, syphilis, and toxemia, both autogenous and exogenous. Brown-Séquard pointed out in 1857 that epilepsy may sometimes have its cause in the spinal cord. Peripheral lesions too may give rise to it. Little can be said about the idiopathic variety. Chaslin has endeavored to show that in this form a constant lesion exists—viz. a diffuse sclerosis of the gray matter, a neuroglial overgrowth—but his views have not been corroborated. After all, we can only enumerate causes; we do not know in any case how these act, and we do not know the ultimate pathology. Many writers apply the name "epilepsy" only to the idiopathic form, while others include all apyretic affections characterized by the occurrence of fits, whether of centric or peripheral origin. Brown-Séquard believes that the distinction between the various kinds of convulsions is artificial, and that the correct classification should be based on the knowledge of the cause.

**Etiology.**—The causes are (1) *predisposing*, (2) *exciting* or *determining*. Among the former, which refer particularly to the idiopathic form, are—

(a) *Age*.—The following tables show the early onset in a large majority of cases analyzed by Gowers, Hesse, and Osler:

Age at onset.		Proportion affected.	
		Gowers.	Hesse.
Before 10 . . . . .		422 . . . . .	393
From 11 to 20 . . . . .		665 . . . . .	364
" 21 " 30 . . . . .		224 . . . . .	111
" 31 " 40 . . . . .		87 . . . . .	59
" 41 " 50 . . . . .		31 . . . . .	51
" 51 " 60 . . . . .		16 . . . . .	13
" 61 " 70 . . . . .		4 . . . . .	4
" 71 . . . . .		1 . . . . .	0
Total,		1450	995

Age at onset (Osler).	Number.	Age at onset (Osler).	Number.
1 . . . . .	74	9 . . . . .	17
2 . . . . .	62	10 . . . . .	27
3 . . . . .	51	11 . . . . .	17
4 . . . . .	24	12 . . . . .	18
5 . . . . .	17	13 . . . . .	15
6 . . . . .	18	14 . . . . .	21
7 . . . . .	19	15 . . . . .	34
8 . . . . .	23	Total,	437

(b) *Sex*.—In Gowers' cases 54.6 per cent. were males, 45.4 per cent. females. Under twenty-five years of age males are slightly in the majority; above twenty-five, the reverse is true.

(c) *Heredity*.—Family neuroses are common, but it is decidedly more the exception than the rule to find either parent epileptic. Féré has given us the lineage of 594 epileptics: 70 had epilepsy, 166 were insane, 88 paralyzed, 21 suffered from general paralysis, 72 from hysteria, 73 from senile dementia, 33 from puerperal eclampsia, 61 from chorea. Among

the direct antecedents of these 594 epileptics, 1024 cases of nervous disorder had occurred.

(d) *Alcohol*.—The causal relationship between an abuse of alcohol by the parents and epilepsy seems rather pronounced. Féré says that of 594 epileptics examined by him, 258 had parents who were hard drinkers. Echeverria refers to 572, 257 of which he believed could be traced directly to the abuse of alcohol.

(e) *Syphilis* does not predispose. When it gives rise to changes in the brain and cord, which in turn cause epilepsy, it is in reality a determining cause.

(f) *Eye-strain* is no longer regarded seriously as a predisposing influence.

The **exciting or determining causes** are traumatisms, various morbid conditions of the membranes of the brain or of the brain proper (*e. g.* after hemiplegia), of the spinal cord or membranes, or peripheral irritation (dentition, worms, a cicatrix, an adherent prepuce, etc.). Not a few cases are dependent upon toxic substances in the blood, as in uremia and lead-poisoning. Excessive indulgence in alcohol or over-eating often precipitates the attack. Great emotion, nervous shock (fright), and masturbation are also said to be able to bring it about, though these are rather doubtful causes. There are cases of bradycardia in which epileptiform attacks occur.

**Symptoms.**—**Petit Mal.**—In this condition the majority of cases belong to the following type: The attack begins suddenly; perhaps while talking to the patient his expression suddenly becomes blank, the face pales, the pupils dilate, and he is evidently not conscious. In a moment or two he gathers his scattered senses, picks up the thread of the conversation, and continues to unwind it. Very often he is not cognizant of any lapse of time or has but a vague idea. If carefully watched for, fine clonic movements may be observed in many cases, it may be of the facial muscles or of the hands. Convulsions never occur, the dominant feature being the unconsciousness. On regaining consciousness the patient may act strangely and appear dazed; it is seldom, however, that he falls in attacks of this kind. Occasionally a peculiar dreamy state takes the place of an ordinary attack, or the individual may be the victim of imperative ideas. Falret has described a condition (*épilepsie larvée*) known as masked epilepsy, in which maniacal outbursts or explosions of passion occur.

**Grand Mal or Haut Mal.**—In many cases some subjective symptom precedes the actual attack. In its most specialized form it is termed an *aura*, and includes any phenomenon, motor and sensory, that ushers in an attack. While the aura differs in different cases, it is almost invariably constant in the same case, so that one will have a subjective sensation of sound, another of light, either flashes or colors, etc. There are other signs that occasionally antedate an attack, and which may or may not precede each attack (headache, drowsiness, change of disposition, palpitation, perverted appetite, sexual or other, etc.). Many attacks begin precipitately with absolutely no previous warning. In such cases the patient may or may not utter a piercing sound (*epileptic cry*), falling at the same time, no matter where or in what position he may be. Hence the danger to which epileptics are always subjected. A peculiar onset occurs in the



so-called "*procurive epilepsy*," in which the patient suddenly starts off and runs some distance before the paroxysm begins.

*Paroxysmal Period.*—In many cases, whether preceded by an aura or not, this stage is ushered in by a *spasm* that is tonic in character. The patient falls, perhaps because of the loss of consciousness, though in those cases in which he drops precipitately he is probably thrown by the violence of the spasm. The head is usually extended, the muscles of the larynx and trunk contracted, and hence the epileptic cry and the dyspnea, while the lower limbs are generally extended, the upper semiflexed, and the fingers tightly clenched. This period of rigidity lasts but a few seconds before *clonic convulsions* appear.

Intercurrent contractions vary in different cases from very mild movements to those so severe as to toss the individual about. The face, pale at first, becomes congested, and the jaw works in churning the saliva into a froth; this is blood-tinged when the tongue is bitten. The respiration is jerky, gasping, and there may be a loss of control of the bladder and bowels. In idiopathic cases this stage lasts from one to five or six minutes. The spasms gradually diminish, and without regaining consciousness the patient passes into a deep sleep, immediately preceded in some cases, however, by coma in which the breathing is stertorous. During the sleep, which lasts about an hour, the patient is completely relaxed. On waking he usually appears confused and complains of feeling tired. His limbs may ache for several days.

Occasionally attacks follow one another in quick succession, with no period of consciousness intervening (*status epilepticus*)—a very dangerous condition.

*Post-epileptic phenomena* are variable. The patient may become maniacal, homicidal, or may simply be mentally deficient for a few days, with perhaps some slight speech-disturbance. In the course of time every epileptic's brain-power deteriorates. Paralysis sometimes occurs, is usually transient, and may be unilateral or bilateral.

**Nocturnal Epilepsy.**—In this condition the attacks occur at night, and may be entirely unknown either to the patient or his friends. He complains from time to time of feeling tired on rising in the morning, his limbs and head ache, and he is generally duller than usual; he may even be confused. Such a history is suggestive, and the suspicion is strengthened if in addition he has urinated involuntarily or if blood-spots are found on his pillow.

**Jacksonian epilepsy** is characterized by spasm that is generally local in character; in fact, it is always so in the beginning, though occasionally it may spread and become general. Consciousness is preserved in the milder forms. Tingling or other subjective sensations may precede an attack. They are usually due to some irritation of the motor cortex (tumors, meningitis, softening, trauma, etc.). Subcortical lesions and certain toxic conditions can also give rise to it.

**Diagnosis.**—When a definite history is obtainable the difficulty of the diagnosis is less, particularly if an aura occurs. The attack can be frequently diagnosed from other epileptoid conditions at the time by the explosive onset, the brief tonic and somewhat longer clonic spasm, profound unconsciousness followed by a deep sleep, and when these are pres-

ent by an involuntary passage of urine, frothing at the mouth, and biting of the tongue.

**Differential Diagnosis.**—In *uremia* the state of the urine (catheterize if necessary), and often the odor, serve to differentiate it. It may be impossible to detect fraud, so perfectly is the disease simulated by those anxious to excite pity, judicial or otherwise, or by those whose accomplices rifle the pockets of sympathetic bystanders. Hysteria may also resemble it very closely. Gowers has tabulated the chief differences as follows:

	EPILEPSY.	HYSTEROID.
<i>Apparent cause</i> . . . .	None.	Emotion.
<i>Warning</i> . . . . .	Any, but especially unilateral or epigastric aura.	Palpitation, malaise, choking, bilateral foot-aura.
<i>Onset</i> . . . . .	Always sudden.	Often gradual.
<i>Scream</i> . . . . .	At onset.	During course.
<i>Convulsion</i> . . . . .	Rigidity followed by "jerking;" rarely rigidity alone.	Rigidity or "struggling," throwing about of limbs or head, arching of back.
<i>Biting</i> . . . . .	Tongue.	Lips, hands, or other people or things. Very rare.
<i>Micturition</i> . . . . .	Frequent.	Never.
<i>Defecation</i> . . . . .	Occasional.	Never.
<i>Talking</i> . . . . .	Never.	Frequent.
<i>Duration</i> . . . . .	A few minutes.	More than ten minutes, often much longer.
<i>Restraint necessary</i> . .	To prevent accident.	To control violence.
<i>Termination</i> . . . . .	Spontaneous.	Spontaneous or induced (water, etc.).

**Prognosis.**—Inherited epilepsy very rarely is cured. Brown-Séquard says most emphatically that idiopathic cases are occasionally cured: I would rather suggest that perhaps they get well. Cases that are evidently symptomatic are curable if the cause can be removed. Death is seldom due directly to an attack. Fatal accidents may, however, be caused by an attack.

**Treatment.**—When an aura occurs, advantage may indirectly be taken of it to aid in aborting the attack—viz. by employing the galvanic method, or by constricting the limb in which the aura occurs, or by forcibly moving the head, putting snuff up the nose, ice to the spine, etc., according to the special indication. Every effort should be made to lessen the liability of danger to the patient—first from falling, and secondly, from the violence of the spasms. One may at times be justified in using ether or chloroform by inhalation to control the severity of the convulsions. After loosening the clothing and putting a cork or something between the teeth to prevent biting the tongue, nothing more can be done at the time. Between the attacks special care should be taken to put the system in good condition, and all sources of worry and irritation should be removed as far as possible. Particular attention should be given to the stomach and bowels. The food should be light and easily digestible.

As to *medicinal measures*, the bromids are of the greatest value. The sodium and potassium salts are most commonly employed, the former, as a rule, being better borne by the stomach. They may be given in milk or in one of the medicated waters. Strontium bromid has been

used rather extensively of late, and has yielded excellent results. While idiosyncrasies are met with, it may generally be given in from 15- to 30-gr. doses (0.972-1.944) three or four times a day, and preferably after meals. Each case must be treated according to its special indications. Symptoms of bromism (acne, sore throat, drowsiness, and gastric disturbance) should be carefully guarded against. Should they develop, the dose of bromid must be reduced, and Fowler's solution administered for a few days. H. C. Wood recommends that the latter should be given continuously with the bromids, thereby preventing, or at all events lessening, the liability to bromism. Other remedies sometimes employed are nitroglycerin (hypodermically), cannabis indica, silver nitrate, zinc, borax, chloral, antipyrin, trional, and tetronal. Surgical measures occasionally yield good results, this being particularly true in focal epilepsy. In idiopathic epilepsy removal of the motor cortex has been tried in those cases in which an aura suggested a local origin—*e. g.* in a center for a particular group of muscles. The results have naturally been less encouraging than in Jacksonian epilepsy, though sufficiently so to justify its adoption. W. Alexander of Liverpool has ligated the vertebral artery in 36 cases, his object being to produce anemia of the brain; 19 cases were either cured or permanently benefited. He has also removed the superior cervical sympathetic ganglia of both sides in another group of cases, with 20 per cent. of either cures or improvement. Neither of these measures has ever been widely adopted, however.

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## MIGRAINE.

(*Hemicrania; Sick Headache.*)

**Definition.**—A neurosis characterized by severe attacks of headache, often paroxysmal and more or less periodic, with disturbances of vision and with or without nausea and vomiting.

**Pathology.**—This is profoundly obscure, since no lesion has ever been discovered. By some it is thought to be a vaso-motor disturbance. Arterio-sclerosis has been present in many cases, but this is also frequently met with where no sign of migraine has ever occurred. Very rarely the disease has been observed in some subjects to replace an attack of epilepsy or even to alternate with true epileptic attacks.

**Etiology.**—The condition is frequently hereditary, and in the large majority of the cases that I have seen it has been transmitted by or through the mother. Various other neuroses are common in families subject to this condition. Females are more frequently affected than males, and migraine seems to be associated with diseases peculiar to women, especially menstrual disorders. Among the exciting causes may be mentioned gastric disturbances, dental irritation, naso-pharyngeal diseases (adenoids, etc.), eye-strain, grief, emotion—in short, anything that tends to lower the physical or mental tone occurring in those hereditarily predisposed. Recently attention has been called to auto-intoxication (leukomainic poisoning) as a cause of certain cases.

**Symptoms.**—As a rule, the patient can prognosticate an attack.



In the cases of slow onset he may feel indisposed for some hours before, being languid, with general discomfort and perhaps nausea. In other cases various *subjective sensations* occur, lasting from a few minutes to several hours. Of these, disturbances of vision are most common, such as flashes of light, spectra, visions of animals or weird forms, or scotoma, etc. Auditory sensations are rare, as are those of the other special senses. After these phenomena have existed for some time *headache* supervenes, when, as a rule, they cease. The pain, at least in the beginning, is usually unilateral, as the name suggests, though later it may and often does involve the entire cranium, spreading from a single point of origin—over one eye, for instance. The affected region may be tender to the touch or it may be the seat of numbness or tingling. *Nausea* and *vomiting* commonly occur, with or without vertigo. A brief period of unconsciousness occurs in some cases, and spasmodic movements may also be observed occasionally. This fact is of particular interest, since it serves to support the view that migraine is in some way related to epilepsy, and, as has been stated, attacks of migraine and epilepsy may alternate. Unlike epilepsy, migraine does not tend to impair the mental faculties, no matter how long the patient has been afflicted. During an attack, however, he may have melancholia or be incapacitated mentally and physically for two or three days.

**Course.**—The disease generally begins in early life, and in nearly half of the cases before the fifteenth year, recurring with a certain degree of periodicity until old age, when it often passes away. It may cease in women at the menopause, and in men between the fortieth and fiftieth years.

The **prognosis** is good as far as life is concerned. This disease is incurable, though the condition of the patient may be alleviated.

**Treatment.**—The management of the disease may be considered under two heads: (1) treatment of the attack, and (2) the treatment between the attacks, which necessarily includes prevention. The patient should be put to bed in a slightly darkened room, and all sources of noise and confusion should be removed as far as possible. The attack may be so severe as to justify the use of morphin hypodermically. The coal-tar derivatives have met with most favor, however, as remedial measures, and preferably antipyrin and phenacetin, though their occasional depressing effect should be borne in mind. The following has given excellent results in my hands:

R <sub>x</sub> . Caffein. citrat.,	5ss (2.0);
Phenacetin,	
Sodii bicarb.,	āā. ʒj (4.0);
Pulv. aromat.,	gr. xij (0.777).

M. et ft. chartæ No. xij.

Sig. One every three hours.

Acetanilid may often be substituted for phenacetin with apparent advantage. In twenty-four hours this may be discontinued, and potassium bromid should be given in liq. ammon. acetatis in doses of gr. x to xv (0.648–0.972). Local applications of menthol, or fly-blisters may be employed, or even superficial points may be made with the actual cautery. Electricity is of little avail. The electric *percutteur* or hammer, however,

has given good results. It consists of a hammer driven at great speed by electricity, thus enabling short sharp blows to be directed to the part.

Between the attacks the general health should be carefully looked after. The uric-acid diathesis is common in subjects of migraine. Haig staunchly advocates the use of salicylates in such cases in addition to the dietetic measures. Anemia should be treated by iron in some form, dialyzed or Bland's pill, or, with those who prefer the organic preparations, ferratin or peptomangan may find favor. The bowels should be kept soluble by means of some saline (Hunyadi, etc.), or by the fluid extract of cascara sagrada. The extract of cannabis indica is employed by some over a long period of time, just as the bromids are in epilepsy. It is given in doses of gr.  $\frac{1}{4}$  to  $\frac{1}{2}$  (0.0162–0.0324) two or three times a day, after meals. While exercise and fresh air are admirable adjuvants to any form of treatment, it must not be forgotten that fatigue invites an attack. Proper rest, care and regularity in dieting, and the avoidance of excitement are the chief points to be observed.

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## ACUTE CHOREA.

(*Sydenham's Chorea; St. Vitus's or St. Anthony's Dance.*)

**Definition.**—The type of chorea described by Sydenham is a more or less acute disease, in certain respects resembling an acute infectious process, and by some regarded as such. It has a special predilection for children, and is characterized by involuntary muscular contractions, by usually slight and rarely-marked mental change, by a great liability to endocarditis and a tendency to recurrence, particularly during the spring and fall.

**Pathology.**—No definite lesion can be ascribed to the disease, though, as is usual in such cases, a great variety of lesions have been described. Chief among these is the embolic theory, advanced by Kirkes and subscribed to by Bastian and Hughlings Jackson. Since emboli are not found in every case, however, they cannot be the sole cause. The infectious theory has also been mentioned above, and certain cases unquestionably seem to be due to micro-organismal activity. For instance, the same organism has been found in the brain, heart, and the blood, or in the vegetations on the valves. Many cases develop suddenly, following fright or some other nervous shock, though these can scarcely be included in either of the two classes just mentioned. Acute chorea may also be due to functional instability of the nerve-centers, or it may possibly result from the irritation of antitoxins.

**Etiology.**—(1) *Age.*—By far the greatest number of cases occur before the twentieth year; in fact, the condition is rare after that age, most cases occurring between the tenth and fifteenth years.

*Sex.*—Females are most frequently attacked, and probably in two-thirds of all cases.

*Race.*—Acute chorea is rarely met with except among the white races. Infectious diseases do not seem to have any predisposing influence.

The relationship, however, between chorea and rheumatism is difficult to ascertain. The milder form of this latter trouble can be readily overlooked in young children, but if we include, as Sturges does, all cases with a history of "growing pains," the relation is of course greater. English statistics show a higher percentage with a rheumatic history than those of any other country, probably because rheumatism is more common in that country than elsewhere. In the United States unmistakable rheumatic attacks occur in but a small proportion of choreic patients either before or during the actual attack of chorea. The latter disease may be, as I have repeatedly observed, followed by rheumatism later in life.

*Endocarditis.*—As has already been stated, some observers regard chorea as the result of a cerebral embolus due to fragments of fibrin being washed from the valves and carried into the circulation to the cerebral vessels. Endocarditis occurs in the vast majority of cases of chorea, but as an effect, not a cause.

*Pregnancy.*—Chorea occurring during pregnancy is apt to be severe. It is most prone to develop during the earlier months, and especially in primiparæ. It often assumes the maniacal type.

*Heredity.*—In about 10 per cent. of the cases a history of chorea can be obtained in other members of the family. Children of neurotic stock are more susceptible than those of a normal type, and in such individuals worry, fright, or mental shock from any cause, also the strain of puberty or of education, are very liable to induce an attack. I have several times seen chorea follow attacks of religious fervor in young girls.

The influence of *reflex irritation* is probably much overrated, whether intestinal, genital, or from ocular defects.

**Symptoms.**—The common or ordinary form is frequently met with, particularly at seasonal changes (H. C. Wood), and especially during the spring and fall. Primary attacks may occur at any time, but recurrent outbreaks are most prone to develop in the spring. There is a slight affection manifested by restlessness, disturbed rest at night, and by irregular and spasmodic *muscular movements*, that are most marked in, or entirely confined to, the upper extremities, the head, or the facial muscles; or the condition may be unilateral. More or less *muscular weakness* is present and the patient tires very readily. The child's *disposition* is changed, outbursts of temper being quite common on the slightest provocation. *Fever* is absent in this form unless complications exist, though when severe joint-troubles or endocarditis is present the temperature will rise. *Anemia* is often present, and with it headaches, irritable heart, and hemic murmurs, very rarely giving rise to symptoms. Indeed, in some instances not even a murmur can be heard, though *postmortem* records of cases with a history of chorea show that in most of them the valves are affected. The mitral valve is most commonly involved, the endocarditis usually being of the verrucose type. There are rarely any sensory symptoms, severe pain, at all events, being extremely rare. Little dependence can be placed on complaints of tingling or burning pain, unless it is voluntarily spoken of, for the mental make-up of choreic patients is such that they are apt to dwell upon slight ailments suggested to them through leading questions. The reflexes do not differ from those of normal children.

There is a more severe type of chorea in which the movements are so



pronounced as to incapacitate the patient. The *psychic symptoms* are generally more marked also, and speech may be interfered with to such an extent as to render articulation unintelligible. Such a condition may occur primarily, but it is probably met with more often after one or more mild attacks. The gravest form of chorea is *chorea insaniens*, in which the movements are violent and constant. Speech is much affected, insomnia is marked, and fever and maniacal delirium develop, followed in some cases by a typhoid condition and death.

The **course** is from six to twelve weeks, though the most trifling cases may recover in a month or less. Others persist six months or even more. In about two-fifths of all cases there is more than one attack, while Gowers has reported one case in which there were nine recurrences. Dr. W. Egbert Robertson observed the case of a girl nine years of age in whom the first attack occurred at the age of six. She had four attacks in three years, each one being more pronounced than the former. In one of my own cases two attacks occurred annually—spring and autumn—for two years. A fatal issue is very rare in children, and absolute recovery is the rule. The maniacal form, which usually develops in adults, especially in pregnant women, as previously stated, is more often fatal, though recovery is also the rule in such cases.

**Diagnosis.**—As a rule, this is quite simple. The age of the patient, the mode of onset, and the character of the movements suffice to prevent mistakes, though the condition may be simulated more or less closely by *hereditary ataxia*. Here, however, the existence of other cases in the family, the nystagmus, the peculiar scanning speech, the swaying, almost reeling gait, and the tendency to contractures will usually be sufficient to differentiate. Yet all these symptoms may be slight, and the case may be under observation some time before its real nature is ascertained. The same is true of *cerebral sclerosis*. It is only by a careful study of such cases in their entirety that a differential diagnosis can be made. The movements may be practically the same in both conditions.

It may be well to call attention to the possibility of meeting with a case that has been treated by arsenic to the point of saturation, when the true nature of the trouble will be masked by the arsenic-poisoning and signs of peripheral neuritis. Such a case was presented by Stengel at the Philadelphia Pediatric Society not long ago.

**Treatment.**—This is largely hygienic—a fact that must be strongly dwelt upon in discussing with the parent the management of the patient. The avoidance of all forms of nerve-strain is of the utmost importance, and the amount of school-work and home-study should be carefully inquired into, and all excess absolutely prohibited. In the milder forms rest in bed is not imperative, but active exercise must be forbidden, since it invites cardiac troubles, the tendency to heart-involvement already being great in chorea. In the more severe forms rest in bed is a *sine quâ non*. In any case an abundance of rest is called for, and when insomnia is present it should be promptly handled. An important element of the treatment that is sometimes indispensable is the change of environment, associated with rest. As a rule, the patients do best in a warm climate and at the seashore. The bromids should first be tried internally, and a warm bath administered just before bedtime. In other cases morphin or chloral may be required, though it must not be

forgotten that the latter is a cardiac depressant; chloralamid or sulfonal should therefore be given the preference. Trional has also given good results in my hands. The bowels must be regulated, and the diet should be light and wholesome, with an abundance of fruit and fresh vegetables. When anemia is present, it is to be met by the use of some one of the preparations of iron. Should reflex irritation be found to exist, it should be corrected at once. Of the therapeutics of the disease not much can be said. We have no specific, but the two most useful drugs are arsenic and cimicifuga, the former as Fowler's solution, and the latter as the fluid extract. Fowler's solution should be given in 3- to 5-drop doses three times a day for a few days, and then increased 1 drop *per diem* until the point of tolerance is reached or the physiologic action is manifest. The remedy is now to be continued, but in reduced dosage. The late Hiram Corson first warmly recommended cimicifuga in the treatment of chorea, and I have found the combined use of this drug and arsenic to be more prompt and efficient than the latter alone, particularly in protracted cases. Other drugs have been employed, but with meager results; among these are the zinc and silver salts and belladonna. Antipyrin has also been used a great deal of late, but with no more promise than is given by the drugs just enumerated.

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## HUNTINGDON'S CHOREA.

(*Chronic Progressive Chorea.*)

**Definition.**—An hereditary disease affecting many members of a family, developing in early adult life, and characterized by irregular muscular contractions, incoördination, and progressive dementia. The disease was first definitely described by Huntingdon in 1872, but other writers had already alluded to it.

**Pathology.**—The changes found consist of chronic pachy- and leptomeningitis, chronic hemorrhagic encephalitis, characterized by round-cell infiltration of the cortex, degeneration of the ganglion-cells, proliferation of the neuroglia, sclerosis of the blood-vessels with dilatation of the perivascular and lymph-spaces, and numerous hemorrhagic foci (Facklam). There are also atrophy of the brain, slight irregular changes in the spinal cord, and multiplication of the nuclei in the muscles.

**Etiology.**—The disease is strictly hereditary, and has been traced through five generations. The offspring of parents that escape are forever immune. It occasionally alternates with idiocy, epilepsy, and various degenerative conditions. It appears to be endemic in certain localities, and still exists upon the southern shore of Long Island, where Huntingdon first observed it.

**Symptomatology.**—The *choreiform movements* commence insidiously and only in the rarest cases become pronounced. They are usually susceptible to voluntary inhibition and disappear during sleep. They usually commence in one extremity and then gradually invade the other parts of the body. There is considerable *incoördination* of move-

ment. The *mental symptoms* consist of progressive dementia, irritability often attaining maniacal violence, delusions of persecution, and rapid diminution of intelligence. In some cases the mental changes are very slight.

The **course** is steadily progressive, but the patient may live to an advanced age.

**Diagnosis.**—The only condition likely to cause confusion is senile chorea with dementia. In this the mental symptoms are usually slight and the motor symptoms more violent. The family character of the disease is also lacking.

**Treatment** is entirely symptomatic.

## RHYTHMIC CHOREA.

(*Hysteric Chorea.*)

In this condition rhythmic choreic movements occur, and affect any of the special groups of muscles or the single muscles. It may be confined to the abdominal muscles (*salaam convulsions*), or, as in a case under my care at present, it may involve only the sterno-cleido-mastoid muscle.

The **diagnosis** is readily assured by the rhythmic character of the movements occurring in an hysteric temperament.

## CHOREIFORM DISORDERS.

### PARAMYOCLONUS MULTIPLEX.

**Definition.**—This is a disease of unknown pathology, first described by Friedreich, and, as its name implies, characterized by clonic contractions in various groups of muscles.

Its **etiology** is obscure. Heredity unquestionably plays an important part, nearly all the patients having among their ancestry cases of one or more forms of nervous disease. It usually develops in early adult life, and is probably more common in males. In one case that I observed it was associated with idiocy.

The **symptoms** of the disease consist of *clonic contractions*, occurring chiefly in the muscles of the extremities and the trunk and only occasionally involving the muscles of the face. These contractions are very sudden; so much so that they have been described as lightning-like. Voluntary movement diminishes them somewhat, emotional disturbance increases them considerably, while during sleep they disappear. The power of the muscles, their size and nutrition, remain unimpaired. The *electric reactions* are normal, but electric stimuli and any cutaneous irritation are apt to precipitate an attack. The *tendon-reflexes* are increased. Sometimes the patient gives vent to a peculiar *grunt*, which is probably due to involvement of the larynx and diaphragm. In some of the cases sensitive points have been found over the spinal column, and not a few have presented other stigmata of hysteria.



The **differential diagnosis** is rather difficult, as it is necessary to distinguish the disease from *chorea electrica* and the *maladie des tics* of De la Tourette; between it and the former there is really no essential difference. *Chorea electrica* occurs in children, and the spasms are perhaps less affected by voluntary movement and more apt to be rhythmic. In the *maladie des tics* the movements are more coördinated, and usually are a repetition of some reflex or voluntary action. Moreover, the patient is apt to emit more or less intelligible sounds. If there is any real difference from clonic convulsions occurring in hysteria, it is only in the presence of hysteric stigmata in the latter and the increase in the muscular contractions under observation. The disease usually begins insidiously.

In the non-hysteric form the **prognosis** is serious, very few of the cases ever showing permanent improvement.

The **treatment** consists of rest, hypodermic injections of morphin, and the application of electricity. The latter seems most effective when applied to the spinal column, a constant galvanic stream being employed and the anode being placed over the sensitive vertebræ. It is not unlikely, however, that these cases are of an hysteric nature.

#### CHOREA ELECTRICA.

**Chorea electrica** is the name applied to at least two, and probably three, varieties of spasmodic disease. The **first**, chronologically, is that of Bergeron. The disease occurs most frequently in children between the ages of seven and fourteen years.

Its *causes* seem to be heredity, anemia, and fright. In some cases dilatation of the stomach has been observed, and it is believed that it bears somewhat the same etiologic relation to this disease that it does to tetany.

*Symptoms.*—It usually commences rather suddenly, and is characterized by lightning-like *contractions* in the muscles of one extremity, although occasionally it may affect the whole body. These are somewhat rhythmic and painless, and do not affect the nutrition of the muscles. They are slightly increased if the patient attempts to inhibit them, but disappear entirely during sleep. Occasionally the respiratory muscles are involved, causing peculiar sounds. Ordinarily the mind of the patient is unaffected; in some cases, however, there is slight melancholia or anxiety during the attack.

The *diagnosis* consists in differentiating the condition from paramyoclonus multiplex and the *maladie des tics*.

The *prognosis* is extremely favorable.

The *treatment* consists in the administration of arsenic and the correction of the anemia and the gastro-intestinal disorders if any exist.

A **second** form, very similar to, if not identical with, this, has been described by Henoch. It occurs at the same age, and is characterized by the same symptoms, with the difference that the contractions are usually localized in the muscles of the back and shoulder, and are almost identical with those produced by a moderate induced current. Usually one side of the body is more affected than the other.

The *etiology* appears to be that of chorea, and cases have occurred after acute articular rheumatism and fright.

The *prognosis* is somewhat more severe than in the form described by Bergeron.

*Treatment*.—Arsenic is without effect, but occasionally the bromids are useful. Henoeh himself, however, prefers a prolonged treatment with the galvanic current, which he has seen produce cure.

A **third** disease, closely allied by its symptoms with the two preceding, but probably of very different etiology, is the *chorea electrica of Dubini*, a disease endemic in Northern Italy. It occurs at all ages, affects both sexes, and appears to be of an infectious nature. Occasionally congestion of the meninges has been found; in other cases there are inflammatory lesions in other parts of the body, and particularly in the lungs.

*Symptoms*.—The disease commences with severe pains in the head, the neck, and the lumbar region. After a brief interval contractions occur in the muscles, usually appearing first in the upper extremities, but rapidly becoming general. They are almost continuous, and are separated by approximately equal intervals, so that they are distinctly rhythmic in character. From time to time there are attacks of general convulsions, that may occur as often as four times per day, and are usually followed by paresis of the limbs. There is slight hyperesthesia of the skin, and usually more or less fever.

The *prognosis* is extremely unfavorable, death occurring in 90 per cent. of the cases. The duration of the disease varies from two or three days to four or five months, death usually occurring from heart-failure while the patient is comatose.

No satisfactory *treatment* has been suggested.

#### FIBRILLARY CHOREA (*Morvan*).

This is an affection characterized by fibrillary contractions, appearing first in the muscles of the buttocks and in the posterior portion of the thigh; it may subsequently extend to the muscles of the body, and even to those of the upper extremities, but never affects the muscles of the neck and the face. It occurs between the ages of sixteen and twenty-two years, and affects chiefly males. Occasionally it follows excessive fatigue. According to Morvan, the lesion is situated in the anterior cornua, commencing usually in that governing the sciatic nerve, and extending upward for variable distances.

The *symptoms* consist of irregular contractions of bands of fibers in the muscle, giving rise to slight irregular tremors and disappearing upon voluntary movement. Occasionally there is a slightly increased secretion of sweat in the affected members, and the skin may become somewhat pinker than normal.

The *diagnosis* is easy, though the disease may occasionally be confused with the lightest forms of paramyoclonus multiplex.

The *prognosis* is favorable, though relapses sometimes occur.

The *treatment* consists in the administration of tonics.

#### ATHETOSIS.

Chorea affecting one side of the body may precede or follow an attack of hemiplegia. In the *former* case the choreic movements usually com-

mence two or three days before the apoplectic attack ; they affect the side that is subsequently to be paralyzed, may be more or less severe, and are usually most pronounced in the arm. As soon as paralysis has occurred they disappear. *Post-hemiplegic chorea*, on the other hand, comes on some days or weeks after the attack, and is usually permanent.

**Athetosis**, a condition first described and named by Hammond, is closely allied to post-hemiplegic chorea. It may be either partial, affecting one side of the body and following a distinct focal lesion of the brain in some part of the motor tract, or else general, in which case it is called idiopathic, and is often found in cases in which a subsequent post-mortem examination fails to reveal any lesion.

**Pathology.**—The local lesions causing athetosis are particularly those of the *optic thalamus*. It is also the commonest sequel of cerebral infantile palsy. These cases usually follow a circumscribed encephalitis, but may also be due to thrombosis or embolism. In any event, the subsequent condition of the brain is usually that of porencephalia, and it may also follow congenital porencephalic conditions.

The *etiology* of the idiopathic or general form is not so well known. It sometimes develops in children born after prolonged and difficult labor, particularly when instruments have been used. It may be associated with microcephalia, idiocy, or paralyzes.

The *symptoms* of the disease consist of peculiar *worm-like movements*, most marked in the fingers and toes, and then in the muscles of the arm and leg, but often also involving the face. The movements of the fingers are very characteristic, and seem to be produced by the combined action of the interosseous muscles and the extensors of the fingers ; as a result, the latter are bent at the metacarpo-phalangeal joints, whilst all the phalangeal joints are extended, in old cases, as a result of constant pulling, usually somewhat over-extended. At the same time the fingers are in constant irregular rhythmic movement. The arm is swung to and fro, bent and straightened at the elbow, pronated and rotated. The movements of the toes are analogous to those of the fingers. In the muscles of the face contractions and relaxations occur, giving rise to peculiar grimaces and somewhat interfering with speech. In those cases in which athetosis develops after a lesion of the optic thalamus there is often associated hemianesthesia ; in those following cerebral infantile palsy, sensibility is intact. The muscles show no trophic disturbances, and, indeed, may be slightly hypertrophied as a result of constant exercise. The idiopathic form is usually bilateral ; the *gait* is curious and characteristic, the patients appearing to be constantly on the point of falling, although they seldom do fall, and maintain their equilibrium almost by a miracle ; whilst the arms swing violently and the face exhibits a series of grimaces.

The *differential diagnosis* of the disease must be made from hemiplegic chorea, into which, according to Leube, it may sometimes pass, but the character of the movements in the two conditions is very distinct. It is sometimes more difficult to distinguish athetoid movements limited to the face from the facial tic or facial chorea. In such cases a history of some infantile cerebral injury will point to athetosis, and the character of the movements should also be considered.

The *prognosis* is hopeless as regards cure.



*Treatment.*—Improvement may sometimes be obtained in the idiopathic cases by the administration of arsenic or the bromids. Hammond has employed nerve-stretching with asserted good results.

#### HABIT SPASM (*Gowers*).

This is not regarded as a true form of chorea, though the affection is nevertheless characterized by localized contractile movements. We owe to Dr. S. Weir Mitchell the first accurate description of “habit-spasm.” It occurs with great frequency in girls from seven to fourteen years of age. General ill-health (a neuropathic state) seems to precede the development of the disease in most cases, though by no means in all. Among specific *causes* may be mentioned overwork at school, fright, eye-defects (refraction-errors—De Schweinitz), nasal obstructions, and the occurrence of the disease in the parents or other older members of a family.

**Symptoms.**—In its commonest form there may be seen *spasmodic movements* of certain muscles of the face (grimaces), particularly vigorous winking or twitching of one side of the mouth or of the cervical muscles, associated frequently with a quick toss of the head to one or the other side, and sometimes with a peculiar sniff.

Less frequently the choreic movements manifest themselves in a shrug of the shoulder or a jerky, sobbing, irregular respiration, occasionally accompanied by a laryngeal sound—a cough or bark. Some authors mention strange tricks that are practised by these children, and Osler<sup>1</sup> speaks of a boy at his clinic who was in the habit every few moments of putting the middle finger into the mouth, biting it, and at the same time pressing his nose with the fore finger.

The **prognosis** in the immense majority of cases is eminently favorable, recovery ensuing within three or four months; but in a small contingent the condition lasts throughout life, usually as a localized muscular spasm (grimace), despite treatment.

The **treatment** embraces two important steps: the removal of all discoverable causes, for which a most careful search must be instituted; and the betterment of the general physical and psychic condition.

In subjects whose health is impaired, efforts to restore the normal vigor of the constitution by fresh-air exercise, cold baths with friction of the body-surface, abundance of suitable food, massage, and the administration of hematinics, particularly iron, are to be assiduously carried forward. Arsenic has been advised, and in one of my own cases proved curative; it is to be employed in augmenting doses in the same manner as in acute chorea (*vide* p. 1147).

#### GENERAL TIC.

(*Maladie des Tics convulsifs; Maladie de Gilles de la Tourette.*)

**Definition.**—A disease apparently psychic in nature and characterized by coördinated spasmodic movements, explosive sounds or words, and imperative ideas, without intellectual disturbance.

The **pathology** of the disease is unknown. It occurs in those suffering from neuropathic heredity, and usually indirectly. It most fre-

<sup>1</sup> *Text-Book of Medicine*, p. 996.

quently commences in childhood—that is, before puberty—and affects either sex. The exciting cause is usually some emotional disturbance.

**Symptoms.**—The disease generally commences in the *orbicularis palpebrarum*, the first movement being an uncontrollable winking. This, as a rule, is rapidly associated with movements of the muscles of the face, causing the patient to exhibit various *grimaces*; finally, other muscles of the body may be involved, and the patient is compelled to repeat many times some apparently purposeful and coördinated movement, as the brushing away of insects or the stroking of the beard. From time to time he emits sounds that may be either inarticulate cries or imitation of some animal, as the crowing of the cock or the barking of the dog, or the repetition of some obscene word (*coprolalia*). These movements are partially under the control of the will, and are diminished by occupation, but increased by emotion. At other times the patient is compelled to imitate sounds that he has just heard, no matter how unusual or unexpected (*echolalia*). A more curious symptom is the imitation of movements that he has observed (*echokinesis*), which may lead to most absurd or painful results. I heard of one man who threw himself violently to the ground when he saw another man fall; and of another having killed his baby, which he held in his arms, by throwing it violently away from him in imitation of a man handling fagots. Still another psychic symptom is the occasional development of imperative ideas. These usually take the form of a desire to recall some unimportant word or syllable (*onomatomania*) or the performance of difficult problems in mental arithmetic (*arithmomania*). One of my patients, a boy of fourteen years, before undertaking a definite act would repeat the words “ten, ten, ten,” three times, followed by a rapid count of figures from one to ten. If riding in a public conveyance, he would do the same, endeavoring to finish before reaching a definite place, as a street-corner, or before hearing the sound of the voice or whistle of the conductor if in a trolley-car. A failure to accomplish the task was cause for intense mental worry. These patients are usually affected at the same time with a certain degree of melancholia or anxiety that interferes to some extent with their normal life. The disease is, as a rule, very obstinate, and ordinarily continues throughout life.

The **differential diagnosis** is usually rather difficult. From *chorea* it may be distinguished by the coördinated character of the movements and by the presence of coprolalia, also by the fact that the movements can usually be controlled partially by the will; from hysteria by the absence of stigmata and the presence of coprolalia; from paramyoclonus multiplex it may be known by the coördinated character of the movements and the absence of increased reflexes.

The **prognosis** is unfavorable for cure; death, however, almost never occurs as the result of the disease.

The **treatment** is symptomatic, and consists in putting the patient in the most favorable physical condition possible; also hydrotherapy, change of climate, tonics, and the correction of any atonic condition are all useful measures. Potassium bromid to a certain extent controls the paroxysms when they become very severe.

## SALTATORIC SPASM.

*(Jumpers; Latah; Palmus.)*

**Definition.**—This is a term applied to a peculiar clonic contraction occurring in the lower legs of a patient on attempting to stand upright. The disease was first described by Bamberger. It appears to occur more frequently in men than in women, and usually in individuals who have suffered from other functional diseases. Occasionally it appears in those who exhibit hysteric phenomena. The condition may develop after severe exertion, and sometimes appears during convalescence from an acute disease. In one of my own cases the condition suddenly arose in the course of habit-chorea. Saltatoric spasm is not a clinical variety of true chorea.

**Symptoms.**—When the patient attempts to stand violent clonic convulsions take place in the muscles of the legs, particularly of the calves. These may cause the patient simply to rise on his toes, or they may be so severe as to cause him to spring from the ground, in which case he usually falls. As soon as he lies down the spasms disappear, but they may be produced in patients lying in bed by pressing against the feet.

The **prognosis** is generally favorable. The attacks usually last for a period of from two days (Gowers) to a few weeks, but a few cases have been recorded that persisted throughout life. Gowers recommends diaphoretic treatment. Antispasmodics may also be employed, and in those cases with hysteric stigmata suggestion is useful.

## CHOREA MAJOR.

*(Pandemic Chorea.)*

This is a form of hysteria (*hysteric chorea*). As early as the Middle Ages it prevailed as a pandemic affection, and was definitely traceable to religious ardor, excitement, or feeling. Writers upon the subject frequently instance an outbreak that occurred among the pioneer settlers in Kentucky. The convulsive movements are general, violent, and continuous; they are aggravated by attempts at voluntary control.

The **diagnosis** rests chiefly upon the causal religious excitement, the character of the convulsions, and the associated hysteric manifestations.

The **treatment** is wholly mental and moral, with the occasional use of drugs to produce quiet and sleep.

## PARALYSIS AGITANS.

*(Shaking Palsy; Parkinson's Disease.)*

**Definition.**—A chronic disease characterized by a tremor; by the peculiar character of the speech and gait, and by a progressive, but very seldom complete, loss of power.

**Pathology.**—Lesions that are probably only senile in type have been frequently described. There are peri- and endarteritis, irregular degenerations in the posterior columns, and numerous amyloid bodies. Other observers have noted changes in the motor cells of the cerebral cortex.

**Etiology.**—Paralysis agitans is a disease of adult life, developing in



the large majority of cases between the fortieth and forty-fifth years ; it is met with more often in men than in women. No definite etiologic factor is known, though, as with most, if not all, nervous diseases, it is predisposed to by mental strain, worry, or trouble of any kind.

**Symptoms.**—The first evidence of the disease is the *tremor*, slight at first, and in the extremities, the hand usually being the first to betray it. The movement is very characteristic, the thumb and fore finger being approximated as in the act of making a pill. At the same time the hand is semi-rotated and the forearm trembles more or less as a whole. The upper arm is either but slightly or not at all affected. The legs are also but slightly implicated. The tremor is most noticeable when the patient is sitting with one leg crossed over the other, the foot then being sure to be in more or less constant motion. When the head is involved (rather the exception than the rule) the motion is a nodding one. The tremors cease when the patient sleeps, but are continuous during waking hours, though it is not rare to meet with cases in which, during purposeful acts necessitating the use of the affected parts, the tremors diminish or even cease temporarily, to return as soon as the voluntary motion is completed. The latter movements, it will be noticed, are awkward, and as the disease advances they become more and more stiff. This *rigidity*, with its consequent impairment of activity, is another cardinal feature of the disease. The patient's movements are slow and apparently measured. There is some impairment of power also, but it is slight, and may be rather from disease than from a direct nerve- or muscle-involvement.

Two of the most striking symptoms of the disorder are the *gait* and *attitude* of the patient. He walks with head and body bent forward, eyes directed toward the ground a short distance ahead, and takes short, mincing, and somewhat hurried steps (*festination*), giving one the impression that he is about to fall, which he would do but for each successive step, which, as it were, gives him a fresh center of gravity. His station is equally striking. The head and back are bent forward, the feet are kept some little distance apart, and one in front of the other, while the arms are slightly flexed and pendulous. From time to time the patient will make a slight forward movement (*propulsion*), or else, if walking, bend or fall backward (*retropulsion*). The facial muscles are set, the eyebrows arched, and the whole expression is "mask-like." The general slow character of all movements, except walking, which is necessarily quicker, is imparted to the speech, though after a sentence is begun the balance may be rendered normally or even hurriedly. The voice may be high-pitched. There are no trophic or sensory symptoms, and the reflexes are normal. Apart from the diffidence, amounting in some cases to a positive dislike for meeting people, and the melancholia occasionally induced by brooding over the affliction, there are no mental changes.

**Course.**—The disease is almost always of slow onset and of insidious progress. Very rarely the earlier symptoms may develop somewhat rapidly, but in every case their further progress is slow. Disappearance of tremor has been observed in the side affected by a subsequent hemiplegia. The course may be interrupted from time to time; even seeming improvement may take place, but it is not maintained. The disease lasts for years, and the patient usually dies of some intercurrent disease.

The **diagnosis** is not at all difficult when the tremor, attitude, gait,

and rigidity have developed. During the earlier stages it may be confounded with *multiple sclerosis*, though this condition develops earlier, and the volitional character of the tremor, the nystagmus, and the scanning speech should serve to differentiate it.

**Treatment.**—The medical management of the disease is absolutely without avail. No drug has been found to exercise the slightest influence on its course. Graduated exercise, however, tepid baths, and massage should be employed to keep up the tone of the muscles.

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### OTHER FORMS OF TREMOR.

1. **Hereditary tremor** has been described by C. L. Dana, who has also reported interesting cases. It may commence in infancy and continue till old age, unaccompanied by detectable lesions.

2. **Simple tremor**, lasting a longer or shorter period (oftener it is comparatively brief), is a rare condition and without serious possibilities. Its etiology is unknown, though it is sometimes aggravated by nervous shocks and other debilitating conditions.

3. **Senile Tremor.**—This is common in old persons, and rarely appears before three-score-and-ten years. It is excited by muscular motions, is always fine, and affects chiefly the hands and arms; more rarely the neck is also involved, and the head may then be seen to tremble.

4. **Toxic Tremor.**—This results from the action of alcohol, lead, mercury, tobacco, and other poisonous substances. I recently saw a case that followed the prolonged use of arsenic for an anemic condition.

5. **Smoker's tremor** is a recognized variety. The tremulousness produced by alcohol, lead, and opium will be considered in the discussion of poisoning from these substances (*vide* The Intoxications).

6. **Hysteric tremor** will receive more elaborate mention elsewhere (*vide* Hysteria, p. 1164).

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### TETANY.

**Definition.**—A disease of unknown cause, characterized by paroxysms of tonic cramp that usually affect the flexor muscles of the extremities, by sensory disturbances, and by a peculiar alteration of the electric reaction of the muscles.

**Pathology.**—Distinct morbid lesions of the nervous system have not been found in all cases. Slight vascular changes in the brain and cord and vacuolization of the ganglion-cells have been described, but these are not peculiar to this disease. It has been supposed that changes ought to be found in the motor nerves, but the most careful observers have failed to detect them.

**Etiology.**—Tetany may occur in epidemics, and has, therefore, been supposed to be infectious. There is some doubt, however, whether these

epidemics are cases of true tetany or are hysteric in nature. There is also some evidence that it is due to an intoxication occurring in the course of some other morbid condition. Tetany is frequently associated with infectious diseases; it also occurs in connection with gastro-intestinal disorders, especially dilatation of the stomach, diarrhea, and intestinal parasites, during pregnancy and lactation, and it is associated with the myxedema that develops after the removal of the thyroid gland. Exposure to cold has often preceded the disease. Occupation seems to exert a remarkable influence upon the predisposition to it, the great majority of those affected being shoemakers or tailors. In childhood males are far more frequently attacked than females, but in adult life this proportion is reversed. Heredity may have some influence, since tetany often occurs in several members of the same family. It is much more common in the winter months, and, curiously enough, it appears to be endemic in certain localities, particularly Leipsic and Vienna.

The **symptoms** fall naturally into two groups: (1) Those of the paroxysm, and (2) Those of the interval. (1) The **first symptoms** of an attack usually consist of peculiar sensory disturbances in the limbs, either tingling, formication, pain, or numbness, and these may precede the attack for some hours or even days. Stiffness of the muscles usually begins in the fingers. There may be slight clonic movements at first, but this is not frequent. The limbs are symmetrically involved. The *spasm* commences first in the hand, the fingers being straightened and flexed upon the hand, and bunched so that the hand has been likened to that of the scrivener or obstetrician. The spasm then extends to the muscles of the forearm and arm, and usually also to the feet and lower limbs. If the cramp is slight, the pain may be insignificant, but ordinarily it is severe, and is increased by attempts to overcome the contractures. The muscles are hard, painful upon pressure, and occasionally fibrillary twitchings may be observed. There is sometimes a slight edema, and often sweating of the limbs. The paroxysms may last for several minutes or for several hours or even days, and may even persist during sleep. If, however, the period is very long, remissions are usually observed. Sometimes a series of paroxysms may occur with considerable regularity. Attacks are more likely to occur at night, and they may also be brought on by prolonged and severe muscular effort, or by emotional shocks. Besides the paresthesiæ in the affected extremities, the patient may suffer from severe headache or pain in the back and neck.

(2) The **symptoms of the interval** are—*Trousseau's sign*—i. e. the possibility of causing an attack by prolonged pressure upon the main nerve-trunks or vessels of the limbs. Fränkel-Hochwart has shown that pressure upon the nerves is essential; pressure upon the vessels acting secondarily if at all. *Chvostek's sign* is a peculiar excitability of the muscles of the face, so that spasms are produced if the trunks of the facial nerve are lightly percussed by a hammer. This occurs in other conditions, particularly the cachexiæ, but in most cases of tetany the spasm occurs if the skin of the face is lightly stroked; and this reaction appears to be pathognomonic. *Erb's sign* consists of a greatly increased electric excitability of the muscles, and, occasionally, of an alteration of the electric reaction, so that ACIC may be greater than KCIC. Moreover, AOTe is often obtained, and, in at least 2 cases, KOTe has also been noted. The



last two reactions occur in no other condition. *Hoffmann's sign* consists of an increased reaction of the sensory nerves to electric stimuli. The facies of the patient is peculiar and characteristic. The face is slightly swollen, dusky, and expressionless, but if carefully examined usually no edema can be detected. Often this swelling is also found in the hands and feet, and may be associated with distinct enlargement of the veins. Even during the interval the feet when at rest have a tendency, particularly in children, to assume a slightly inverted and extended position.

The *sensory disturbances* are of considerable importance. Often in the affected limbs there may be slight diminution in sensibility. In addition to this, there are changes that can only be described as true hysteric stigmata—*i. e.* hemianesthesia, or symmetric areas of insensibility to pain or touch. Often there are points of special sensitiveness on the spinal column, and in some cases pressure upon these will precipitate an attack. Distinct *trophic changes* have also been observed, consisting of herpetic eruptions along the course of the nerve-trunks, falling out of the hair, and sometimes even painless ulceration. The contractures are not invariably limited to the extremities. Sometimes the muscles of the neck, back, and larynx are involved; sometimes also the diaphragm, and occasionally the compressor urethræ. Involvement of the larynx gives rise to stridulous respiration; involvement of the diaphragm to severe dyspnea; when the urethra is compressed there is retention of the urine. *Fever* occurs in about one-half the cases; it is slight and generally limited to the paroxysm. The urine usually contains a large amount of phosphates, and less frequently indican is present in excess.

The **differential diagnosis** is very easy, as a rule, if the patient is seen during a paroxysm. The severer forms may, however, be confounded with *tetanus* or *meningitis*. Tetany can be distinguished from the former by the fact that the spasm begins at the periphery and rarely affects the muscles of the jaw. From the latter it may be diagnosed by the absence of coma and the slighter degree of fever. The diagnosis from certain forms of *ergotism* is more difficult, and can often be made only by careful attention to the etiology. The *hysteric forms* cannot always be differentiated, for, as has been stated above, hysteric stigmata occur in true tetany; but an epidemic occurring among young women should give rise to a suspicion of the true nature of the malady.

**Prognosis.**—The duration varies from a few days to many months, the most obstinate forms being those due to thyroidectomy and chronic diarrhea. The disease cannot be said to have disappeared until the characteristic symptoms of the interval (Trousseau's, Erb's, and Chvostek's signs) can no longer be elicited. The prognosis is usually favorable, nearly all cases tending to spontaneous cure. Death, however, may occur from chronic diarrhea, from respiratory failure when the diaphragm is involved, and from cachexia strumipriva.

The **treatment** is purely symptomatic. The patient should be placed in the most favorable hygienic conditions and given plenty of nourishing food. During the spasm bromids or chloroform-inhalations seem to give the best results. The most important therapeutic measure is the correction of the underlying cause. Thus, in children rickets is almost invariably associated with tetany, and the most efficient remedies are iron and cod-liver oil. Intestinal disorders should be treated according to the

principles laid down in the discussion of these diseases. The form due to removal of the thyroid gland always disappears under a course of thyroid-medication, while that occurring during pregnancy usually persists until delivery.

## INFANTILE CONVULSIONS.

(*Eclampsia Infantilis.*)

UNDER this term are grouped a number of conditions, with convulsive attacks as the common symptoms.

**Pathology.**—The pathologic changes may be divided into two groups: (1) those bearing an etiologic relation to the convulsive attacks, and (2) those that are merely consecutive. Among the former are meningeal bleeding, tumor, gliosis (either hypertrophic or atrophic), and hydrocephalus. Then there are general conditions that seem to predispose to this condition or, at any rate, are frequently associated with it, such as rachitis. The consecutive lesions are hemorrhages into the meninges or into the substance of the brain and the spinal cord, an increase in the amount of cerebro-spinal fluid, and congestion of the pia or the substance of the brain.

The **causes** are: 1. Organic brain lesions. 2. Neuropathic tendency, that is manifested later as hysteria or epilepsy. 3. Emotional disturbances, as fright. 4. Rickets, in about 30 per cent. of all cases. 5. Acute infectious disease, especially as an initial symptom of pneumonia, and more rarely of scarlet fever, small-pox, and pernicious malarial infection. 6. Inflammation of the serous membranes, as meningitis, where the relation is direct, or pleuritis or peritonitis. 7. Kidney disease, in which they are uremic. 8. Peripheral irritation; dentition has long been supposed to be a chief factor in their causation, but it is now believed that the chief cause is the presence of rickets. Intestinal parasites have also been found, particularly the *ascaris lumbricoides*, and the convulsions have ceased after their expulsion. 9. Debility, especially that resulting from gastro-intestinal disorders.

The **symptoms** of the attack vary according to its intensity. In the most severe form they resemble in all respects those of an epileptic seizure. At first the eyes deviate upward or to one or the other side, and the gaze becomes fixed and staring; next there are *twitchings of the muscles of the face*, sometimes slight and limited to one side, and sometimes general, often involving the muscles of mastication and giving rise to trismus or gnashing of the teeth. Next there are *tetanic contractions* of the extremities, the fingers being strongly flexed, the hands flexed upon the arms, and the feet in the position of *pes equinus* or sometimes in the dorsal flexion, and both legs and arms rigidly extended. Often the muscles of the trunk are involved, and there is either *opisthotonos* or respiratory cramp, with excessive hardness of the abdominal muscles. This rigid condition is interrupted at brief intervals by sudden twitchings, or occasionally the convulsion becomes *clonic* instead of *tonic*, and there are repeated extensions and contractions of the extremities, shaking of the head, and quivering of the whole body. As a result of the respiratory cramp,

*cyanosis* rapidly develops and may reach an extreme degree. The forced respirations give rise to a foam that collects about the lips, and is often mixed with blood from the bitten tongue. Urine is often, and feces occasionally, passed involuntarily. In nearly all cases unconsciousness is complete. Many of the slight attacks are accompanied by a cry or by an attack of screaming. The tetanic state usually lasts for a minute or two; then there are a few clonic movements, relaxation becoming rapidly complete, and the spasm is ended by a few deep respirations. The child may return to consciousness, although it is usually drowsy or stupid, or it may pass into a deep sleep from which it cannot be aroused. Often in the latter condition attacks will recur at irregular intervals, and sometimes a single attack may continue for some time, although from time to time there are slight twitchings followed by partial relaxation (*status eclampticus*—Lewis). The attack may come on suddenly, or, as is more frequently the case, it may be preceded by a period of restlessness and irritability. A milder form of the spasm consists of sudden fixation of the eyes, slight twitching of the body, and a peculiar dusky pallor that passes away in a few moments. In other rare cases consciousness may persist, although the patient is aphasic. Laryngismus stridulus is an analogous condition (*vide* Diseases of the Larynx, p. 477).

The **diagnosis** of the condition is very easy. The recognition of the cause, however, is very important and often difficult. Every case should be first examined for rickets, and then the gums should be investigated; also the condition of the child's nutrition and the presence of symptoms of gastric or enteric irritations. If fever exists, it is important to discover its cause. The character of the convulsion is often of value in distinguishing between the idiopathic or reflex type and that due to organic brain-disease. Convulsions beginning immediately after birth, or an injury, either persisting or else disappearing gradually, are probably caused by meningeal hemorrhage. An attack of a Jacksonian type would, of course, indicate the presence of a focal lesion; and if this be a tumor, there will probably be bulging of the anterior fontanel, severe headache, and the ophthalmoscope will reveal a neuro-retinitis. If, after the attack, pareses or paralyses are present, a focal lesion is still more likely. Hydrocephalus is usually recognized with ease. Some cases exist, however, in which it is impossible to discover any adequate cause.

The **prognosis** varies according to the etiology. In cases with organic brain-disease it is unfavorable as regards cure. In those forms that precede epilepsy or functional nervous diseases the spasms usually disappear after the first dentition, and the patients appear to have recovered for a time. In those, however, in whom the symptoms are due to some peripheral irritation or to rachitis, the outlook is fair, although even these now and then develop into permanent epilepsy. The convulsions themselves are either often immediately fatal, or so exhausting to the patient that he succumbs readily to the disease that produced them. In these cases the prognosis depends upon the frequency and severity of the attacks, death usually terminating those in which the status eclampticus has been established. The prognosis for ultimate cure depends also in part upon the length of time that the condition has existed; if but for a short time before an arrest has been established, recurrence is much less



likely. Gowers, however, says that even after a year's duration permanent cure may sometimes be obtained.

The **treatment** naturally falls into two parts—that of the attack and that of the interval. Unquestionably, the most efficacious antispasmodic that we possess for this condition is chloroform. A few drops may be put upon a handkerchief and held carefully over the nose and mouth of the little patient. A very small quantity usually suffices, and the effect is almost instantaneous. In addition to this, chloral and the bromids may be given by the rectum, and it is often useful to add to these one of the coal-tar antipyretics, particularly antipyrin. Morphin may be given hypodermically. Formerly hot mustard-baths were much in favor, but unless they do good at once they are not likely to be of any use. In a very obstinate case under my care they were absolutely valueless, and were replaced by momentary immersion in ice-cold baths and vigorous friction, which seemed to act very favorably. If any known source of irritation is present, as an overloaded stomach, it should be relieved at once, if possible, by the stomach-tube or an emetic. An enteritis may be temporarily benefited by an enema or by a moderate dose of calomel. The treatment during the interval depends upon the nature of the cause. If rachitis exists, it should be treated according to the principles laid down in my discussion of this disease. If dentition is suspected, the gums may be lanced, but this should only be done when they present distinct signs of irritation. Gastro-intestinal disorders of any kind should be relieved as soon as possible, and intestinal parasites must be expelled. In infectious diseases the convulsions usually disappear after the initial stages, and require no further attention. In organic brain-disease, providing it be not syphilitic in nature, very little can be done. Finally, in those cases in which no cause can be discovered bromids are the only resource, and should be given in sufficient doses: from gr. iij-v (0.194 to 0.324) per day to children of six months, and from gr. v to x (0.324 to 0.648) to those between six and sixteen months.

## OCCUPATION-NEUROSES.

**Definition.**—Conditions in which the performance of certain habitual coördinated movements is prevented by the development of cramp, tremor, paralysis, or pain. The commonest form is *writers' cramp* (graphospasmus, mogigraphia, scriveners' palsy).

The **pathology** of this condition is unknown. It is probably purely functional, and the discovery of appreciable lesions is not to be expected, though nodular thickening of the peripheral nerves has been described in a few cases.

The **etiology** is various. Males are far more frequently affected than females, the condition usually occurring in early adult life, although children are not exempt. The condition always occurs in those whose occupation demands much writing, and Gowers lays great stress upon improper methods of holding the pen, particularly those in which most of the writing is done from the wrist; that is, with the muscles of the

forearm and hand. As scrivener's palsy occurs sometimes in those that write properly, and as a similar condition is not uncommon in other occupations, it seems unlikely that this is the most important cause. A person with a neurotic temperament is far more apt to be affected by the disease than one with a normal nervous system; we, therefore, frequently find it associated with hysteria, neurasthenia, or great bashfulness, and not infrequently it is possible to elicit a neuropathic heredity in the family history. It is also met with in certain other nervous diseases (epilepsy, locomotor ataxia—in the early stage.) Often the patients admit that at the time the disease developed they were suffering from severe anxiety.

**Symptoms.**—**Motor.**—When the patient attempts to write there is usually a cramp of the flexor muscles of the forearm, so that the pen is held more or less rigidly, and it is almost impossible to control its motions. Less frequently there is a cramp of the extensor muscles, so that the fingers are spread and it is impossible to hold the pen at all. Sometimes there is a sudden twitching, and the pen may be thrown altogether out of the hand. The spasm is nearly always tonic in character, but often it is associated with a fine tremor, and at times there are clonic movements. In some cases, and particularly those occurring in patients showing hysteric stigmata, there is a coarse, irregular tremor, most marked when the patient is under observation. Paresis is frequently associated with the cramp, so that the arm soon becomes tired and it is almost impossible to write. This fatigue may in a few moments progress to almost complete paralysis of the arm, but, curiously enough, both fatigue and paralysis disappear as soon as some coördinated movement other than writing is undertaken.

**Sensory.**—Pain is very common, and is neuralgic or cramp-like in character, being referred either to the muscles, bones, or joints. In intensity it varies from a dull ache to the most excruciating burning, and may form the only symptom, the muscles performing their work perfectly. At times it is sharply localized to one particular joint, affecting either the metacarpal bones or the fingers. Quite often the patient complains of a tingling or burning sensation in the limb, or it may be numb and the hand feels, when writing, as if a heavy weight were attached to it. Often there is tenderness either of the muscles or the nerves, which may be localized in certain points. In very severe cases *vasomotor disturbances* occasionally occur. The disease ordinarily commences slowly. At first the subject notices that the handwriting is not quite as perfect as before, a stroke occasionally going astray; later distinct spasms appear, and these are finally associated with pain.

The **diagnosis** is usually easy. Care must, however, be taken not to call every disturbance of writing writer's cramp; thus in paralysis agitans, in slowly-developing hemiplegia, in multiple sclerosis, and in locomotor ataxia disturbances of writing frequently—in fact, almost invariably—occur. Moreover, those cases in which hysteria or neurasthenia seems to be at the bottom of the trouble should be carefully differentiated from those that are apparently idiopathic.

The **prognosis** is rather unfavorable, though complete cure is sometimes attained.

The **treatment** consists first in a total cessation of writing; if this is impossible, various mechanical devices may be employed to use another

set of muscles or the old ones rather differently, such as a thick penholder or one constructed with supports for the fingers. Local treatment of the arm in the form of electricity should be advised; the anode of a constant galvanic current of medium intensity should be placed over the sensitive points on the nerves and over the bodies of the muscles. Massage, and particularly careful and systematic exercises, are also of great value. At the same time, the general condition of the patient must not be neglected. In cases associated with hysteria, hypnotism may be employed, and in those associated with neurasthenia a treatment appropriate to this condition. If possible the patient should learn to use a type-writer and abandon penmanship altogether. Operative treatment is more likely to do harm than good.

The other occupations in which this disease occurs are—telegraphy, playing on musical instruments, cigarette-rolling, milking, and sewing. Strümpell suggests that stuttering is an analogous affection of speech.

## PERIODIC PARALYSIS.

(*Paroxysmal Familiar Paralysis—Goldflam.*)

**Definition.**—A disease characterized by paroxysmal attacks of complete paralysis, and alteration in the electrical reactions and structure of the muscles, occurring in many members of a family.

**Pathology.**—Excised fragments of muscle show rarefaction of the fibers and slight vacuolation, without multiplication of the nuclei or proliferation of the connective tissue.

**Etiology.**—The disease is purely hereditary. Both sexes are affected. The attacks appear to be more frequent in summer, and often seem to occur after overfilling the stomach.

**Symptoms.**—The attacks are preceded by *prodromes* in the form of vague discomfort or paresthesia. The patient then usually falls asleep and awakens completely paralyzed. Speech, deglutition, and the sphincters are unaffected. During the attack there is often transient *albuminuria*, with blood-cells in the urine. The *reflexes* are abolished. The *paralysis* lasts from twelve hours to three days, and then there is an outbreak of perspiration, with gradual recovery, the muscles of the head first regaining power. During the interval the muscles react slowly to faradism and galvanism, and are easily thrown into CaC tetanus. The muscular changes have been demonstrated in children of an affected family in whom the disease had not yet developed.

**Diagnosis.**—Usually easy. Goldflam was once puzzled by a case of acute ascending paralysis.

**Prognosis.**—The disease does not kill; but there appears to be no tendency to recovery.

**Treatment.**—This involves only caring for the children during the attack. Even persistent starvation does not decrease their frequency.



## HYSTERIA.

**Definition.**—A condition of the general nervous system partaking of the natures of both a neurosis and a psychosis, and characterized by a vast multiplicity of clinical manifestations, all indicative of a loss of voluntary control over the inhibitory will-powers.

**Pathology.**—Hysteria is to be regarded as essentially a morbid entity, without, however, any tangible pathologic features. The most careful *postmortem* examinations of subjects who have while in life manifested pronounced hysteric symptoms have failed to reveal any organic nervous alterations, however slight. The occurrence of the affection in men as well as in women excludes the former theory of a uterine pathology, which, though an idle fancy, held sway for so many centuries and gave origin to the name by which the condition is generally recognized.

**Etiology.**—There are a large number of predisposing and exciting factors, all of which, however, may be grouped under a few dominant heads. Thus among the former must be mentioned, pre-eminently, *heredity*. The investigations of many neurologists and alienists of divers lands have gone far to demonstrate that at the foundation of the vast majority, if not of all, of the hysterias is to be discovered an inherited neurotic tendency or temperament. The family histories of these patients generally reveal a large number of consanguineous neurotic or hysteric individuals.

The diseased condition in question is apparently in close relationship with the various psychoses and major neuroses (epilepsy, chorea, tetany).

In the process of transmission one generation may entirely escape the pernicious influence, and successive generations may manifest strikingly different evidences of the disease, in one the neurotic and in another the psychic element predominating. A curious phenomenon that is worthy of mention is the apparent *contagiousness* of hysteria; moreover, the baleful influence one neurotic individual exerts over the unfortunates of this temperament explains the so-called “hysteric epidemics” that have swept over communities, and even over vast tracts of land or entire countries, at different periods of the world’s history. Similar, though limited, outbreaks may still be seen in the nervous wards of hospitals or in religious and political conventions, and these depend largely upon the general prevalence of the neurotic disposition untempered by a virile will-power.

The hysteric temperament may be, and often is, fostered by improper and pernicious modes of life, especially by luxurious and sensuous living and by the habit of gratifying every desire of the will during early life. It is manifested at this early stage of the individual’s existence by hypersensitiveness, brilliancy, undue enthusiasm, and a more or less erratic turn of mind.

*Sex* is a strongly predisposing cause of hysteria, and, while it is by no means limited to females, the great majority of instances of the disease occur among girls and women. Rarely, however, instances of a most rebellious nature occur in the opposite sex.

*Age.*—The condition is generally encountered between the ages of fifteen and thirty years. After the latter age the frequency of the disease rapidly diminishes, and prior to the onset of puberty the cases re-

corded are also comparatively few in number, although exceptionally it is seen in infancy.

A very influential factor in the production of the disease is the *lack of proper mental development*. It stands to reason that those who are coarse and illiterate, and who have not been taught the lessons of self-control, and who are subject to the various and multiplex superstitions that are ever prevalent among the masses, will respond more quickly and more generally to the causes that tend to destroy mental equilibrium. Hence, hysteria or insanity shows its rankest development among those whose education and culture are defective. This is, however, by no means an inevitable law, for over-stimulation of the faculties may be just as deleterious as under-stimulation, and some of the brightest lights of the world have manifested at various periods of their lives decided hysteric symptoms.

*Improper hygienic surroundings*, tending as they do to enervation and physical depression, are influential predisposing factors in the evolution of hysteria. In addition to poor and insufficient food, lack of proper ventilation, overcrowding in foul habitations, and insufficient bathing, must be mentioned the enervating influence of hot and moist climates. It is generally conceded that more cases of hysteria occur in the warm than in the colder portions of the temperate zones, and that this proportion increases *pari passu* with the height of the temperature.

Finally, the causal influence of the chronic toxemias (alcoholism, morphinism, absinthism, saturnism and intoxications by other metals) is to be mentioned. In systemic poisoning the depraved condition of the physical reacts upon the mental organism, and sooner or later hysteric manifestations may be found to coexist with the original toxic phenomena.

The *exciting causes* of hysteria may be grouped as follows:

(1) Most commonly psycho-neurosis follows some profound emotion or mental or moral shock. Thus, in neurotic males it may be excited by excessive and protracted business-worry or excitement, or by active competition in certain lines of occupation, or by some heavy and unexpected monetary reversal. In females it is not uncommon as a sequel to the establishment of puberty and the menstrual function, or to the physiologic arrest of menstruation at the period of the climacteric. Especially is it prone to develop in young and illegitimately pregnant women, or during the first pregnancy in newly-married women of a neurotic temperament. Great religious excitement during the progress of a revival-wave and profound political upheavals have been most potent in establishing the disease in numerous instances; and other profound mental impressions, of fear, grief, or great and unexpected joy, have assumed the exciting rôle.

(2) Extreme physical prostration, the result of some very acute or much protracted chronic disease, may exert an etiologic effect. Thus, some of the most marked and intractable forms of the disease have resulted from the specific fevers (typhoid, typhus, and the other exanthemata), while it is not rare in a varying degree in the final stages of tuberculosis, chronic nephritis, and other grave constitutional affections of long standing.

(3) The so-called "traumatic hysteria" has come to occupy a prom-

inent place in the etiologic category of the disease. Especially do we find the incurable varieties of hysteria resulting from a slight or it may be a more severe traumatism. It must be remembered that a considerable period of time may intervene between the date of the injury and the appearance of the initial hysteric symptoms, so that in all cases it becomes of the utmost importance to make a careful study of the patient's history for signs of traumatism, however remote. It has also been noted that oft-repeated minor traumatisms may essentially result in some hysteric manifestations. So well recognized has this fact become that it is not unusual to discover an hysteric element in certain occupational ailments. Thus the irritable neurasthenic condition of the centro-spinal axis so common among the employees of railroads, and known as the "irritable hysteric" or "railroad spine," is now acknowledged as the type of this traumatic hysteria. In fact, any slight injury or a blow upon the head or a trivial injury of another portion of the body, especially if it be brooded upon, may ultimately cause hysterо-epilepsy.

(4) Finally, in a limited number of cases sexual excesses and masturbation are the influential factors in the production of hysteria. The sexual origin of the disease, which was formerly the chief etiologic theory, has now come to occupy only a minor causative rôle, but the tendency to entirely abolish it as a cause of the disease is as much an error in the opposite direction. These sexual cases, though few in number, do exist, and are especially to be found among the class of so-called sexual perverts.

**Clinical History.**—Clinically, hysteria presents three well-marked stages, known respectively as the *prodromal*, the *convulsive*, and the *latent*. The latter is also designated as the *interconvulsive stage* or the *period of the stigmata*, and during this period the number of the symptoms and their complexity almost baffle attempts at classification; they can, however, best be portrayed by presenting them under the heads of the various systems (*vide infra*).

(1) **The Prodromal Stage.**—The prodromes are invariably present, and at times they are more marked than at others. They are evident alike to both patient and physician, and are largely psychic in nature. There is noted a marked mental depression associated with introspection, and, it may be, with a form of mild mania or of melancholia. A condition of aprosexia develops, and the patient becomes irritable, restless, and discontented. The mental derangement may manifest itself in the form of delusions or nightmare, and there is a characteristic neglect of the toilet and attire. Naturally, there result disturbances of the gastro-intestinal tract—viz. anorexia, nausea, vomiting, constipation, and perversions of taste. The patient is pale, and the expression assumes an anxious or, it may be, vacant cast. These phenomena persist for several days, and are followed by emotional disturbances—spasms of hysteric laughing and crying—that immediately precede the *aura*, which is as marked a feature in hysteria as in epilepsy. It may assume one of a number of forms, but more commonly it has an ovarian, a cervical, a cerebral, or a surface or cutaneous origin (unilateral). Very frequently the convulsion is preceded by a condition of extreme sensitiveness and pain in one or both ovarian regions, so that the lightest touch at a point on the abdominal surface one inch above Poupart's ligament, and midway



between the pubis and the anterior superior iliac spine, will elicit exquisite tenderness. This is so constant and characteristic that many patients can invariably predict the onset of the convulsion. Not infrequently the aura begins in the neck, the patient experiencing a sensation as of a ball lodging in the throat (*globus hystericus*): this is due to a spasmodic contraction of the muscles of the pharynx and esophagus, and is accompanied by tachycardia and a sense of suffocation. If the aura originate above the scalp, it is characterized by the sudden appearance, generally in the top of the head, of a severe neuralgic pain, as if produced by the entrance of a nail (*clavus hystericus*); this is frequently associated with vertigo and tinnitus aurium. The aura, finally, may appear in the form of spots of cutaneous tenderness, mainly localized upon the trunk, to which areas has been given the name of *hystero-genous zones*.

(2) **The Hysteric Convulsion.**—Closely following upon the footsteps of these prodromes, and immediately following the aura, the hysteric convulsion may appear. Most commonly this is epileptoid in nature; rarely it assumes a less common type. Hence it becomes necessary to describe several of the forms of the convulsions—viz. (a) the *epileptoid* (hystero-epilepsy); (b) the *gymnastic* (clownism); (c) the *emotional cataleptic*, or *dramatic*; and (d) the *delirious*. All of these forms may be present in the same attack, the one passing quickly into the other, or, as in the abortive cases, one or the other form will predominate. Briefly described, the characteristic features are as follows:

(a) *Epileptoid (Hystero-epilepsy).*—Immediately upon the appearance of the aura the patient commonly emits a shriek and falls upon the floor or in some convenient place, taking special care to do herself no injury: this is in strong contradistinction to the true epileptic spasm. The head and limbs are thrown about by more or less violent clonic muscular spasms, and at times a condition of opisthotonos or other trunkal contortion (emprosthotonos, pleurosthotonos) may be noticed; these muscular movements, however, are more or less volitional, and are not the aimless movements of the true epileptic. The patient may or may not foam at the mouth. There is a constant twitching of the eyelids and the eyes are rolled about, but apparently retain a more or less observant expression. Consciousness, as a rule, is not fully lost. The facial muscles are distorted, rapid changes of expression being noted (*hysteric trismus*), and respiration is somewhat impeded. As the convulsion passes off the movements gradually subside, and the patient generally sinks into a state of quiescence or, it may be, into a light sleep. This may be followed by complete temporary recovery, or the epileptoid may pass into one of the other forms of the convulsive seizure. The duration of the spasm as described is usually longer than that of a true epileptic seizure.

(b) *The Gymnastic Form (Clownism).*—This stage is characterized by violent and grotesque muscular movements. Here are to be grouped all of the more curious manifestations of the disease recorded in the history of medicine. The most difficult feats of the contortionist are performed with apparent ease; the patient may suddenly begin to dance or jump at a most astonishing rate, persisting in the movements until she drops from pure physical exhaustion. The so-called religious ceremonies of the Shakers of Lebanon, Pennsylvania, and of the Jumpers of the Middle

Ages, are manifestations of this form of hysteria. In children the attack may appear as the so-called *beast-mimicry*, in which the movements or sounds of the lower animals may be simulated; such is also the explanation of the condition known as *spurious hydrophobia*. *Eclampsia nutans*, the "nodding spasm" or "salaam convulsion" of young children, is likewise a sub-variety of this form of the disease. Consciousness is never lost during this period.

(c) *The Emotional Cataleptic, or Dramatic Form*.—In this form the patient seems to suffer from delusions or hallucinations that are apparently the outcome of the preceding condition. The emotion that is most developed in the patient's moral constitution now dominates his spasmodic actions. As Lloyd aptly expresses it: "The third period of the hysteric convulsion is one of dramatic representation of emotional images, and these are of countless varieties, according to time and person." All of the manifestations of the cataleptic state are present. Sensation is largely abolished, consciousness is retained, and the patient is usually able to recall events that have transpired during the period. Especially common now is the assumption of dramatic and passionate attitudes, which, as described by Richer, include "the attitude of the cross, of defence, of menace, of appeal, of lubricity, of ecstasy, of dread of animals (as rats), of scorn," and the like. The body of the patient retains, at times for indefinite periods, whatever position is first assumed (*hysteric catalepsy*). In some cases the patient falls into a condition of apparent sleep or narcolepsy (*hysteric sleep*, *hysteric somnolence*, *hysteric trance*) of varying degrees of intensity; this may persist for any period of time, from a hour or two up to weeks, months, or even years. In these extreme cases, while the patient at first appears to be in a normal sleep, sooner or later the body assumes a corpse-like appearance, with pale, waxy skin, almost imperceptible respiration and cardiac action, and a subnormal temperature.

(d) *The Stage of Delirium*.—The final stage of the hysteric convulsion is but a continuation of the preceding period, with, however, a cessation of the muscular movement to a great extent. The tendency now is to delirium of a mild type, tinged with more or less melancholia. Consciousness is maintained throughout this stage, and there now appear some curious motor phenomena that may persist for days or weeks. These may consist in the abolishment of muscular power in various portions of the body. Very often associated with these motor phenomena is noted a condition of mutism that lasts for indefinite periods of time.

*Hysteric paralyses* occur, and may simulate any form of the organic paralyses (monoplegia, hemiplegia, paraplegia). In many cases the patient is left with a more or less permanent spasm of a single set of muscles or of associated sets. These so-called *hysteric contractions* may affect any portion of the body. One arm may be bent at the elbow or one leg at the knee; in the former case the fingers are rigidly contracted and embrace the thumb, which is crossed upon the palm, while in the latter the toes are strongly flexed upon the plantar surface and the foot is inverted. The ankle- and knee-jerk persist. In other cases a curious spastic gait is produced that closely simulates that of spinal sclerosis. The muscles of the hips, shoulder, back, and neck (*hysteric torticollis*) may share in the process. In women the muscles of the diaphragm and abdominal walls may be involved (*hysteric pseudo-cyesis*). *Hysteric rotatory spasm*, *hys-*

*teric athetosis*, and *hysteric tremor* are all dependent upon a spasmodic action of the muscles affected. The convulsive seizure generally is of *short duration*, lasting but fifteen to thirty minutes. Occasionally, however, there is developed a prolonged convulsive status, during which time the patient continually falls from one convulsion into another, until one hundred or more may be recorded and the excess of nervous power is exhausted.

(3) **The Latent or Interconvulsive Stage, or Period of the Stigmata.**—After the convulsive attack the patient enters upon a more or less prolonged interval of comparative quiet; this is characterized, however, by numberless and varied phenomena—the *hysteric stigmata*. The whole course of the affection may be comprised in this period, convulsions being absent. As I have already stated, these can best be described under the heads of the various systems:

(a) **The Nervous System.**—This presents the most characteristic hysteric stigmata. They are generally grouped into the three classes of *motor*, *sensory*, and *psychic*.

The *motor symptoms* have already been referred to in part in the description of the hysteric convulsion. They embrace every variety of muscular pathology, from obdurate paralysis to and including tremor, incoördination, and tonic spasm or contraction. The hysteric paralyses, as stated, may be absolute or partial, and either general or limited to groups or to individual muscles, and may simulate any variety of true paralysis of organic origin. There is usually noted an exaggeration of the reflexes of the affected side, muscular wasting may or may not be present, and it is not at all uncommon to find associated contractures and sensory phenomena. The paralyzed limb or limbs show evidences of circulatory disturbances, as edema and bluish discoloration. In the paraplegic cases it is unusual for trophic disturbances (bed-sores) to appear; some atrophy of the affected muscles may, however, be noted. Hysteric tremors are not infrequent, and are usually well marked and persistent. They are generally associated with contractures and other hysteric stigmata.

Hysteric incoördination (*hysteric ataxia*) has also been termed *astasia-abasia*; it is one of the rarest of the motor phenomena of hysteria. The name implies an inability to stand or walk, although muscular power in the legs and trunk is retained (*vide Astasia-abasia*, p. 1181). *Hysteric contractures* may occur as distinct phenomena or may be associated with some form of hysteric paralysis. Usually the contractures occur with startling abruptness, and are most intense and persistent, resisting even the effects of sleep and the anesthetics. There may be associated sensory phenomena. The toes and the fingers are most frequently the seat of contracture, but the muscles of the face and neck may likewise share in the affection.

**Sensory Symptoms.**—The anesthetic, hysteric, and paresthetic varieties are noted. Patients showing hysteric anesthesia are able to run pins into themselves without showing the slightest degree of suffering. The anesthesia may be general or it may involve but half of the body or scattered areas of the cutaneous surface. *Segmental anesthesia* is the term applied to that condition in which a limb or a portion of a limb is involved. Not only is the skin affected, but often the deeper tissues as



well, and there is generally some vasomotor involvement, as is shown by the fact that punctures by a needle are not followed by bleeding. There is often associated an anesthesia of one or more of the special senses (*hysteria amaurosis* or *blindness*, *hysteria deafness*, and *hysteria anosmia*). The conjunctivæ very frequently escape. The anesthesia is severe, as a rule, immediately after an hysterical convulsion, but it may be entirely absent throughout a given case of hysteria. There is often contraction of the field of vision or inversion of the color-fields, the red being more extensive than the blue.

*Hysteria hyperesthesia* is also a frequent clinical manifestation, and is generally confined to limited areas, as the ovarian, mammary, or spinal regions, or to one of the larger joints (*hysteria joint*), simulating organic disease of the part. These conditions can be recognized by etherizing the patient, when perfect mobility of the affected joint is noted. When one of the mammae is involved, the organ becomes exceedingly painful to the touch and slightly edematous (*hysteria breast*). *Hysteria paresthesiæ* include the common varieties of formication, dead fingers, and the like.

**Psychic Symptoms.**—These have already been mentioned among the prodromal symptoms—violent and capricious changes of temper, mental depression and unrest, melancholia, and a notable lack of volitional power whereby the patient becomes especially open to the suggestions of the hypnotist. Such patients may develop into that strange condition known as “double consciousness.”

(b) **The Digestive System.**—Among the usual clinical manifestations of this group may be mentioned *anorexia* (which may be complete), a strange and persistent perversion of taste, occasional uncontrollable vomiting without nausea (*hysteria vomiting*, *anorexia nervosa*), marked dyspepsia, and at times extreme emaciation with dryness and a parchent-like feel of the skin. Excessive flatulence and the peristaltic unrest of Küssmaul may be marked symptoms, as may also either diarrhea or constipation. *Hysteria hematemesis* is the result of swallowing blood; this is usually drawn from the gums or tonsils, or it may be taken secretly by the patient from other external sources.

(c) **The Respiratory System.**—Difficulty of respiration (*hysteria dyspnea*) is not uncommon, and is characterized by an extreme rapidity and shallowness of the respiratory movements. These are much out of proportion to the heart-beats, and are unassociated with cyanosis. In other cases the disturbance assumes the form of uncontrollable yawning, sneezing, or hiccoughing, due probably to a spasmodic action of the involuntary muscles of the bronchial tubes and diaphragm. *Hysteria cough* is a troublesome, and very often a stubborn symptom, occurring especially in young females. It is dry and barking, and, as a rule, unaccompanied by expectoration. At times it may be followed by *hysteria hemoptysis*, in which there is an escape of light-red fluid from the pharyngeal mucosa. *Hysteria aphonia* is also frequently noted; in this condition the patient scarcely speaks in an audible whisper. In such cases restoration of the voice is as of sudden occurrence as is its loss. In one of my own cases aphonia manifested almost true intermittence for a period of five years, while during the last two years or over it has stubbornly persisted even without remission.

(d) **The Vascular System.**—*Hysteric tachycardia* is often noted, and much less frequently *hysteric bradycardia* appears. A variety of *pseudo-angina* is not of rare occurrence (*vide* Angina Pectoris, p. 671). Very frequently the patient exhibits a localized flushing of the skin (*hysteric erythema*), and especially of the face and neck, or, as has already been noted, there may be an apparent bloodlessness of a part. Profuse general or localized sweating is not uncommon, and may at times be bloody.

*Hysteric fever* may be mentioned here as a rare manifestation, the bodily temperature usually being normal in hysteria. The elevation of temperature may be moderate or there may be an extreme hyperpyrexia ( $110^{\circ}$ – $120^{\circ}$  F.— $43.3^{\circ}$ – $48.8^{\circ}$  C.), without grave results. If this be associated with localized neuralgia, it becomes a difficult matter to diagnose between the neurotic condition and organic disease of the apparently affected part.

(e) **The Urinary System.**—An excessive flow of urine (*hysteric polyuria*) is of very common occurrence, while the opposite condition (*anuria*) is much rarer.

**Diagnosis.**—The diagnosis of hysteria must depend largely upon the history of the attack, the previous history of the patient, and the recognition of the features of the neurotic temperament. Individuals possessing this habit are generally spare of body, of quick movements, brilliant and changeable eyes, an emotional disposition, and unstable mental equilibrium. Close and judicious inquiry among the friends will, as a rule, elicit a history of previous hysteric seizures of laughing and crying. The great tendency of the profession, unfortunately, is to overlook true organic conditions and ascribe the patient's symptoms purely to an hysteric attack. Moreover, it must not be forgotten that even genuine hysterics may develop true organic disease, either during an attack of hysteria or in the intervals, and these intercurrent affections may, though rarely, terminate fatally.

**Differential Diagnosis.**—Very important is it to distinguish between hysteric and true paralyses, and between hysteric and organic abdominal tumors. In the following tables the most striking points of difference between these conditions have been set down:

HYSTERIC PALSIES.	ORGANIC PALSIES.
Occur without a previous history of organic disease, but with a neurotic history. Traumatism may be the cause.	Are always secondary to organic spinal or brain-disease or to traumatism.
Are accompanied by other hysteric stigmata or perversions of sensation.	Hysteric stigmata are absent.
Are not accompanied invariably by wasting of the muscles involved.	Are always accompanied, sooner or later, by muscle-wasting.
Reactions of degeneration are absent.	Reactions of degeneration are present.
The paralysis is apt to be more or less transient and shifting.	The paralysis is permanent and marked.
The power of motion returns before sensation.	Sensation first reappears.
In hysteric hemiplegia the facial muscles are not involved.	The facial muscles of the same or opposite side are always involved in true hemiplegia.
Anesthesia generally causes relaxation of hysteric contractions.	Organic paralytic contractions are not affected by anesthesia.

## HYSTERIC ABDOMINAL TUMORS (PSEUDOCYESIS).

Almost invariably occur in neurotic women near the menopause.  
 The percussion-note is invariably tympanitic.  
 Anesthesia causes a disappearance of the tumor.  
 Is variable as to size and tonicity.  
 Is accompanied by tympany and flatulence.

## ORGANIC ABDOMINAL TUMORS.

Occur irrespective of sex.  
 The percussion-note over the swelling is dull, or a dull tympany.  
 Anesthesia has no effect upon the tumor.  
 Slowly but steadily progresses in size.  
 The bowels are not always distended by gas.

The differential diagnosis between hysteria and true neurasthenia, and epilepsy, will be found in the discussion of the latter two affections.

**Prognosis.**—As regards death, the prognosis in hysteria is good; true hysteric patients never die of the disease, nor does the hysteric spasm ever result fatally. As to an ultimate cure, however, the prognosis is very doubtful. If the disease occur early in life and if there is a marked congenital neurotic tendency manifested in the patient, there is almost no hope of effecting a permanent cure. In the acquired cases, under proper moral and hygienic control great benefit may be effected or even an absolute cure recorded.

**Treatment.**—**Of the Temperament.**—Accurately speaking, the treatment of hysteria should be begun before birth. Neurotic women bearing children should be subjected to a course of rest-cure and mental and moral suasion, and the condition of their nervous systems should receive the careful attention of the attending physician. Neurotic children require the greatest care during the developmental period. A strong physique must be secured by proper attention to out-of-door exercise, and, for the time being, even at the expense of mental culture. Such children should not be subjected to the "cramming" process so common in our modern courses of education, but should be trained, if possible, at home, where the element of competition may be eliminated. Systematic hours of study and of recreation (with absolute rest from study during the summer months), and opportunities of travel and change of air and scene, will work wonders in these hyperesthetic little individuals. Especially at the time of puberty is the greatest of care required in order to avoid an additional strain upon the already seriously taxed nervous system. In addition to the foregoing a strict watch must be kept over the moral nature of the child. The satisfaction of every whim and the lack of moral suasion are the surest ways to develop the hysteric temperament. When possible the child should be taken away from the enervating influences of city life. The diet should be plain, but nutritious, and all over-indulgence is to be absolutely prohibited. Frequent bathing and friction of the skin are very beneficial, as well as careful regulation of the emunctories generally.

**The Hysteric Convulsion.**—During the hysteric seizure the patient must be carefully watched, and suitable measures should be adopted to cut short the attack; all extreme measures, and severity, however, are unwarrantable. Cold plunge-bathing, the dashing of cold water into the face, or the hypodermic injection of apomorphin, thereby producing a profound mental shock, may have a beneficial effect. Pressure over the



ovary or upon one of the large vessels (as the carotid) will sometimes promptly induce a termination of the attack.

**Internal Treatment.**—In this period of the disease it is probable that most can be done to improve the condition of the patient. In addition to the general laws of mental and physical regimen already advanced, the patient should be taught, as far as is possible, the undignified condition into which she is sinking, and advised and encouraged to exert powerful efforts to control her nervous organism. To this end she should also be given full doses of the nerve-sedatives and antispasmodics (valerian, asa-fetida, sumbul, musk, and camphor), together with the general tonics (iron, arsenic, strychnin). I have repeatedly found the rest-cure of Weir Mitchell especially beneficial at this time; it is fully described under *Neurasthenia* (*vide* p. 1177).

Hypnotism has commanded considerable attention during this stage of the disease, and it is claimed that under the suggestion of the hypnotist an absolute cure very frequently follows. This is not altogether true, however, for while many patients are undoubtedly benefited by this procedure, the good result must be attributed not alone to the suggestion of the operator, but also to the profound mental effect produced upon the patient by the mysterious process.

In the treatment of the organic manifestations, which, it must be remembered, are dependent entirely upon the general nervous condition, the physician is called upon to exercise the greatest amount of tact. As far as is possible the mind of the patient must be directed away from the affected part. The *irritable bladder* must be treated by internal remedies, as boric or benzoic acid, salol, or the compound infusion of buchu, and not by local irrigation and catheterization.

*Hysteric vomiting* may not require any special medication. Occasionally, however, it may be relieved by rectal alimentation. Cocain hydrochlorate in the form of a 10 per cent. solution (dose internally), and the application of mild counter-irritation or of a small fly-blister over the epigastrium will be useful. Cannabis indica, acetanilid, phenacetin, and antipyrin, in small doses and only when absolutely needful, will relieve *hysteric neuralgias*, especially the cephalalgia. For the pseudo-angina pectoris, digitalis, strophanthus, caffein, amyl nitrite, or nitroglycerin, or a combination of these drugs in suitable doses, may be exhibited.

For the *pelvic hyperesthesia* of hysteric females local applications (tincture of iodine, croton oil, or a small fly-blister) over the ovarian region may prove very beneficial.

*Hysteric palsies*, either general or local, and hysteric disturbances of the special senses, must be treated on general principles. As far as is possible the patient's attention must be directed from the affected part or parts, and an occasional local blistering, the use of galvanism and massage, with daily friction, will be of service, especially when they are supplemented by an appropriate course of internal medication.

## NEURASTHENIA.

**Definition.**—Functional exhaustion and irritability of the nerve-centers. Neurasthenia is the expression of an abnormal sensitiveness (irritability) in response to stimuli, and of weakness of the nerve-centers presiding over the organic functions. Several varieties—cerebral, spinal, cardiac, and gastric—have been distinguished, owing to the fact that the predominating features may be manifested by single organs or systems of the body. That the disease is essentially generalized in all instances, however, I do not doubt. It is not a psychosis.

**Pathology.**—A variable degree of weakness of the sympathetic centers, permitting congestions on trivial provocation, is obvious, but there are no discoverable lesions (coarse) in the nerve-centers that are peculiar to the affection. C. Y. Hodge<sup>1</sup> has invited attention to certain changes in nerve-cells during the active exercise of their function, and something of pathologic importance has been added to our previous knowledge by his observations. There are many causes and associated affections that present a variety of morbid lesions, but they are purely incidental. It should be pointed out here that neurasthenia is often found in association with other functional nervous disorders—a fact that has not only caused mental confusion among certain authors, but has also led to the belief among others that as a distinct affection it does not exist.

**Etiology.**—The causes are divisible into—1, predisposing; and 2, exciting. Among the **former** (*a*) *heredity* heads the list. A clear history of nervousness or morbid irritability in one or both parents (oftener the father) is at times obtainable. Ancestors that were sufferers from gout, rheumatism, syphilis, tuberculosis, and chronic alcoholism, all diseases that exhaust vitality, may have transmitted to their offspring a strong neurasthenic disposition. The latter have inherited a small stock of nervous energy with which to begin life's unceasing struggle.

Of other predisposing factors may be mentioned in particular (*b*) improper training, mental and physical, and (*c*) the character of the mental pursuits, those entailing strains being especially deleterious. (*d*) *Age* and *sex* are not without appreciable effect, most cases occurring between the twentieth and fiftieth years, when the work and worry of life are maximal; they are more frequent in men than in women.

**Exciting Causes.**—According to my own observations, *traumatism* has an active potency, though it is probably not the most frequent cause. *Overwork*, at least in America, is responsible for a greater number of cases than any other single factor, and in estimating its effects the relativity of individual nerve-capital must be carefully considered. Associated causes are to be observed in unpleasurable emotional excitement, mental worryment, particularly if dependent upon love-affairs, and sexual excesses. *Abuse of the sexual organs*, excessive venery, masturbation, and the like are powerful in producing neurasthenia. Finally, as stated under Pathology, the condition may be induced by other functional and organic affections (symptomatic neurasthenia).

**Symptoms.**—The subjective symptoms are protean and varied, but these are often learned only after close interrogation, the patient being unduly reticent as a rule. Among the more prominent and numerous

<sup>1</sup> *Journal of Morphology*, vol. v. No. 11, p. 95.

features entering into the symptom-complex of neurasthenia are great *irritability*, *physical fatigue* without adequate reason, even to a feeling of utter exhaustion on rising in the morning, *disturbed sleep*, *headache*, with a sense of weight and constriction, *impairment of memory*, *anorexia*, and *constipation*; the patient is very irritable, dispirited, is fearful, and frequently sinks into a state of absolute dejection. Female sufferers—and less frequently males also—may manifest strong emotions, and in such cases the condition presents many points of resemblance to the milder forms of hysteria. The external appearances may be indicative of sound, vigorous health; oftener, however, the physiognomy is worn and anxious.

The **motor phenomena** include, besides readily oncoming exhaustion of the muscular strength under exercise, a variable condition of the tendon-reactions. On the whole, however, they are increased. Muscular *tremors* (fine) are sometimes present, and particularly when neurasthenia is the result of trauma or fright (Dercum), and spasmodic contractions (usually brief) of small isolated groups of muscular fibers of the face, trunk, or extremities are observed.

The **sensory** disturbances are varied and sometimes striking. The patient makes constant complaint of feeling “tired” or “never rested,” and indeed sometimes betakes himself to bed for this reason. A feeling of “lightness,” giddiness, and even true vertigo, may occur and recur, and rarely the latter symptom is wellnigh continuous. The *headache* (previously mentioned) is often wholly dependent upon mental work, since it disappears with the cessation of the latter. Another form of *pain* is a dull aching that may be generalized, though more commonly it is confined to the small of the back and limbs. *Spinal tenderness*, when sought for, may often be elicited over certain circumscribed areas or mere points, and it may be combined with a deep-seated ache or an exacerbating pain (“spinal irritation”). Cutaneous *hyperesthesia* is common, but *anesthesia* is not found in uncomplicated neurasthenia. Numbness, either spontaneous or as the result of slight pressure, is a conspicuous feature for a variable period upon or near to the nerve-trunks, and linked with it there may be a generalized or localized feeling of coolness of the body-surface, or of pricking sensations (formications) and circumscribed subjective sensations of heat and burning.

The *psychic* symptoms grow out of the same fundamental conditions as do the *physical* symptoms—*i. e.* fatigue of the nerve-centers. As would be expected, then, the capacity for sustained mental work is generally lessened, and the power to concentrate or rivet the attention upon any subject as well. The patient is self-centered, sensitive to a degree, easily angered, and is morbidly suspicious. His emotional nature is unstable, and the mental depression (before mentioned) deepens until it approaches true hypochondria.

*Insomnia*, varying in form, is frequent, and disturbances of the organs of special sense are not wanting. The eye presents the most important fatigue-symptoms. *Vision* may be imperfect (blurred), and continuous close use of the eyes be impossible. There is a lack of power of accommodation and retinal hyperesthesia may supervene. The pupils may be unnaturally large. All forms of *tininitus* constantly arise in neurasthenia, and may lend so vivid a coloring to the clinical picture that the real nature of the attack is liable to be overlooked. I have recently seen a case of the sort



occurring in a clergyman (thirty-six years old), in whom the diagnosis of aural disease had previously been made. This symptom, like all others due to neurasthenia, may, however, be associated with genuine organic diseases of the ear (*otoneurasthenia*). Disturbances of taste also tend to appear, but they are of minor importance. *Vasomotor* disorders, such as hot flushes and profuse sweats, commonly arise in consequence of the diminished tone of the arteries; these form quite distressing fatigue-symptoms. Visible throbbing of the superficial vessels and of the abdominal aorta, and rarely also of the veins and the capillary pulse, occur in the affection (*vide* Aortic Regurgitation, p. 597). The *urinary phenomena* may excite particular attention owing to their prominence, and this remark applies especially to the frequent combination of neurasthenia and lithemia (*lithemic neurasthenia*). Oxaluria and transient glycosuria and albuminuria may also be present. The daily amount of urine is often small, and less frequently it is large. The sexual apparatus is weak and irritable, as shown by frequent seminal emissions (nocturnal) and incomplete erections, and, if the subject be married, by premature ejaculation. The fear of becoming impotent often renders the mental attitude of those really potent such as to excite the keenest compassion. The orgasm in the female and the emission in the male are followed by a sense of prostration and mental depression.

The somatic disturbances referable to the heart (palpitation, precordial pain) have been considered under Neuroses of the Heart, and the various gastro-intestinal features in the discussion of Neuroses of the Stomach. Reference has already been made to several clinical varieties based upon the predominance of special and localized groups of symptoms—*e. g.* when the reigning features are spinal the variety is termed *spinal neurasthenia*; when these are presented by the sexual apparatus, *sexual neurasthenia*; and so on; but I am in entire accord with Dercum when he avers that groups of symptoms cannot be considered as sufficient ground for the division of neurasthenia into separate forms.<sup>1</sup>

**Diagnosis.**—That cases of neurasthenia are misdiagnosed as other conditions, and the reverse, I feel convinced. An important matter at the outset is to avoid confounding the neurasthenic symptoms (secondary) of various local and general organic diseases with the primary form by a careful exclusion of the latter. From *hysteria* the diagnosis is as follows:

#### HYSTERIA.

By nature a psycho-neurosis.  
Occurs in individuals presenting a marked hereditary taint.

The onset is frequently abrupt.  
The clinical features are dependent upon an excess of nervous energy.  
Presents the characteristic stigmata, as paralysis and anesthesia in most cases.  
Is sometimes accompanied by violent convulsive seizures.  
Neuralgic attacks are infrequent or altogether absent.  
Insomnia is not marked.

#### NEURASTHENIA.

A true neurosis.  
Occurs as the result of nerve-tire, overwork, and the like in individuals not necessarily presenting hereditary taint.  
The onset is always gradual.  
Is characterized by a notable lack or insufficiency of nerve-force.  
These are absent.  
Convulsive seizures never occur.  
Neuralgic attacks are very common.  
Insomnia is very common.

<sup>1</sup> *Nervous Diseases by American Authors*, p. 73.

Hysteria, it is to be remembered, may be a complication of neurasthenia, and this association must be distinguished from simple hysteria.

**Prognosis.**—Neurasthenia is a curable disease if appropriate treatment be commenced before secondary structural changes set in and render the use of the most approved measures of no avail. In long-standing cases deleterious habits (morphinism, chloralism, alcoholism) are sometimes developed and prevent the possibility of a cure. Hysteria (the complication) tends to delay, but does not preclude, recovery.

**Treatment.**—The first step should be, after locating the major cause or causes, to remove them, or, if this be impossible, to minimize their baneful influence so far as may be. For example, if the conditions have been induced by overwork of the brain, rest for the organ must be procured; if sexual excesses have been the obvious responsible factor, rest for the sexual apparatus is imperatively demanded. In the next place, the mental and moral environment must be conducive to contentment and to wholesome forms of exercise of the mind. In this way the exhausted stock of nervous energy can be often increased by the natural recuperative forces alone. Indeed, successful removal of the essential etiologic influences is in the milder forms followed by prompt recovery. In not a few instances the symptoms disappear as the result of a prolonged sojourn in a suitable climate or by travel for a considerable period with its ever-accompanying change of scene, though it is well in doing so to avoid the din and excitement of large cities. The compulsory rest and complete isolation, combined with the purity of atmosphere, afforded by a sea-voyage sometimes work admirable results. Unfortunately, many subjects suffering with neurasthenia, and particularly males, are either unable or unwilling to arrest the loss of nervous function by ceasing their excessive activities. In the majority of instances, for the reasons above stated, certain other measures—hygienic and medicinal—are to be advised.

Next to the importance of removing the exciting agents, stands rest. In severe and long-standing cases this should be made as nearly absolute as possible, while in the milder forms merely lengthening the hours for sleep or rest in bed, as first pointed out by Dr. S. Weir Mitchell, often suffices. The amount of rest must be accurately proportioned to the necessity of individual cases. To Dr. S. Weir Mitchell belongs the credit of having introduced the "rest-cure" in the management of this disease. This mode of treatment in very old, and in profound cases produces curative effects unobtainable in any other manner, though it does not give complete restoration to health, as a rule, and must be variously modified in individual instances. It embraces not only absolute rest, but also "passive exercise" and forced feeding. Both body and mind must receive rest; hence confinement often is not sufficient, and the patient must also be strictly isolated from friends and relatives, particularly if hysteria be associated.

The patient is to be put in charge of a properly selected nurse, who will afford agreeable entertainment by suitable conversation and reading under the instructions of the physician. In desperate cases the patient should not be allowed to feed himself, must not rise to void the urine or feces, nor even turn in bed without the help of the nurse.

The neurasthenic also demands a special *dietary*, that is to be made up at first of milk. This should be administered in small quantities at the

beginning (3iv or v—120.0 or 148.0, every two hours), and slowly and gradually increased until at the end of a week or ten days large quantities are taken (3vij-x—236.0—300.0 every two hours). When whole milk cannot be readily digested skim-milk should be employed. To the milk, should the patient become decidedly hungry, may be added in the course of five or six days very light nutrients (plain boiled rice, a soft-boiled egg) and a little later meats (lamb-chop, steak). Constipation calls for the use of unbolted bread (graham), fresh and stewed fruits, and butter. This simple dietary is to be enriched until three large meals are taken daily—"such, for instance, as a breakfast of fruit, cracked wheat, one or two soft-boiled eggs, or a good-sized steak, well-served chops, bread and butter, and milk; a dinner of a good slice of roast beef, with vegetables and boiled rice (in place of potatoes). The supper should remain as a light meal of bread, butter, fruit, light pudding, and milk" (Dercum). Tea, coffee, and alcohol should be avoided.

*Passive exercise, massage, and electricity* form an essential part of the "rest-cure," though the former should not be commenced until the second or third day. At first it should be continued for a few minutes only, and consists of gentle rubbing or light strokes. As tolerance becomes established massage should be practised for a longer period (about an hour). Deeper rolling, kneading, and spiral manipulations are then allowable. The direction of the venous blood-current—toward the center of the body from the periphery—is to be borne in mind, and all massage-motions are to be made in the same direction. This measure is to be carried out by the nurse, who should be a well-trained masseuse and thoroughly acquainted with the details of her work. Electricity, like massage, compensates for the lack of exercise. The slowly-interrupted faradic current is to be selected, and the aim should always be to induce satisfactory contractions with the least amount of pain. The current should be applied to the individual muscles, one of the extremities being selected, and the poles applied over the motor points, passing from muscle to muscle until all have been faradized. The time of each sitting should not exceed half an hour. The entire body should also receive the faradic current (rapidly interrupted). A large sponge moistened with salt water is applied at the nape of the neck, and another to the soles of the feet, and the strongest current tolerable is thus used. This process should be continued from fifteen to twenty minutes, and, like the faradization of the single muscles, it is to be repeated at intervals of twenty-four hours.

The rest-cure in all of its details should be continued for a period ranging from four to eight weeks. The patient should leave his bed in the most gradual manner, and should sit up for a few minutes only at first, the time being gradually lengthened; soon exercise may be commenced in a like manner and be cautiously increased. During this period of convalescence it is my custom to omit the electric treatment, while the massage is continued at intervals of two or three days for some weeks. After the patient has reached the point of marked improvement, as evidenced by a large appetite, the disappearance of the most pronounced subjective symptoms, and especially by a substantial gain of weight (twenty to twenty-five pounds—11.3 kgms.), he should be advised to make a change of air to the country, or to the mountains or the seashore (if it be not the summer season).



Hydrotherapy is positively and rationally serviceable in the management of neurasthenia. The water may be employed in the form of the shower, spray, bath, or pack, and is most efficacious when quickly applied for a few moments and followed by vigorous towelling to reinforce the action of the cold. A portion of a garden-hose with a sprinkler is readily attached to the water-pipe in the bath-room and furnishes the readiest means of applying cold water. Extreme caution is necessary at the beginning of the application of cold to the surface, since there are neurasthenic subjects that not only fail to receive benefit, but are rendered worse thereby, in consequence of a highly sensitive organization.

Drugs are of minor importance in the treatment, and their routine use is to be condemned. Laxatives are often needful, and in my experience broken doses of calomel, followed by a saline, or the fluid extract of cascara, have proved most effective. For a further consideration of the treatment of the gastro-intestinal symptoms the reader is referred to *Neuroses of the Stomach*.

Strychnin is constantly being employed in the treatment of this affection, but its use should be limited to the more profound types. Full doses are required if we would expect good results. In cases in which anemia is marked arsenic may be employed with advantage. Phosphorus has also been recommended, but it tends to disturb the digestive organs and rarely gives striking general results.

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## ACROMEGALY.

(*Giantism*.)

**Definition.**—A disease first recognized and described by Marie, and characterized by a progressive and peculiar enlargement of the face and extremities.

**Pathology.**—Those cases that have been examined *postmortem* have shown, as the most constant change, an enlargement of the pituitary body, with a corresponding dilatation of the sella turcica, and a persistence of the thymus gland. Less frequently there is fibroid degeneration of the thyroid gland. A few cases, however, have been reported in which one or all of these organs were normal. The lips, tongue, and trachea are usually considerably enlarged, and the sexual organs may either be hypertrophied or atrophied, the latter condition being more common in the uterus and testicles. The bones of the extremities and face are thickened, apparently chiefly as a result of hyperplasia of the spongy portion, and Klebs has shown that the peripheral vessels, particularly those in the affected bones, are also larger. Occasionally there are hypertrophy of the heart and enlargement of the spleen and liver.

The **etiology** of acromegaly is unknown. Marie believes that it is a form of systematic dystrophy analogous to myxedema, and probably due to interference with the function of the pituitary body. Freund holds that it is a sort of inversion of growth associated with alteration in the sexual organs at puberty. Klebs believes that it is due to a neoplastic condition of the vascular tissues, associated with a functional persist-

ence of the thymus gland. Various diseases have preceded the development of acromegaly, but none with sufficient regularity to indicate that the subsequent appearance of the latter condition was other than accidental. Both sexes are about equally affected, and the disease ordinarily commences in adolescence.

The earliest **symptom** is usually an increase in the thickness of the fingers and toes, so that rings, gloves, and shoes are too small and can no longer be worn. This *enlargement* is chiefly in thickness, although there is also a certain amount of increase in length. Both the soft and hard parts are affected. The nails are flattened, longitudinally ridged, and more friable (*spade-like hand*). The *face* becomes considerably enlarged; the supraorbital ridges project, giving rise to a rather simian aspect; the nose becomes broader and longer; the cheek-bones project; but the most positive characteristic is the enormous enlargement of the lower jaw, so that it becomes broader and prognathous, and the lower teeth can no longer be brought in apposition with the upper. The *spinal column* is ordinarily kyphotic, the change affecting the upper dorsal and cervical regions. Frequently there is also an associated scoliosis. The rest of the skeleton remains unaffected for a long time; finally, changes may be observed in the clavicles, sternum, ribs, pelvis, and particularly in the patellæ. The *skin* sometimes shows slight pigmentation; the hair is rough and may become thinner; the *muscles* occasionally exhibit increased electric excitability, and less frequently there is muscular atrophy with reactions of degeneration. The lips, tongue, and tonsils are usually enlarged, and the larynx is increased in dimensions, so that the *voice* becomes deep and rough; this is a very characteristic symptom in women. Ordinarily, an area of dulness can be detected in the upper part of the sternum that has been ascribed to the persistence of the thymus gland. The *tendon-reflexes* may either be normal, diminished, or abolished. They are never exaggerated. The *urine* is increased in amount, and glycosuria is often present. The secretion of *sweat* is also greatly increased. The subjective symptoms consist of severe intermittent or continuous *head-ache* and of a *diminution of the visual power*. There may be paresis of the third nerve, giving rise to external strabismus, and sometimes to *temporal hemianopsia* as a result of pressure upon the central part of the chiasm by an enlarged pituitary body. Sometimes late in the disease there are occasional momentary general tremors. The patients often present polyphagia and polydipsia. Neuro-retinitis and subsequent atrophy of the optic nerve may also occur. Sexual power is usually abolished. In women menstruation ceases early in the disease, and the breasts atrophy. The mental condition is affected, and there are usually great apathy and diffidence (perhaps explicable by their changed appearance), loss of memory, and even, in some cases, imbecility.

**Diagnosis.**—In the later stages the appearance is characteristic, and acromegaly can then hardly be confounded with other diseases. The peculiar enlargement of the extremities, the oval, prognathous, and distorted face, the deep, rough voice, the more or less pronounced pigmentation of the skin, the wasting of the muscles, and the profound cachexia give a perfect clinical picture. In those cases in which the cachexia has become extreme there are from time to time peculiar tremors or spasms of the body.

**Differential Diagnosis.**—In the earlier stages the disease is most easily confounded with the *hypertrophic pulmonary osteo-arthritis of Marie*. In this both hands and feet are greatly enlarged; but the fingers are club-shaped, the face is not involved, and there usually exists some chronic pulmonary complication. In a case that I observed there were bronchiectasis and bronchorrhea. From *osteitis deformans* it may be distinguished by the fact that in this condition chiefly the long bones of the limbs and the flat bones of the skull are hypertrophied and very painful. *Elephantiasis* may be distinguished by the fact that it attacks the lower limbs, does not involve the bones, and the skin presents a granular or a nodular appearance. From *arthritis deformans* acromegaly may be distinguished by the fact that the disease develops late in life and is associated with great deformity of the joints, the face ordinarily escaping. The following table (after Dercum) will serve to distinguish two diseases that are apt to be confounded with one another:

ACROMEGALY.	MYXEDEMA.
Occurs most commonly in early adult life.	A disease of mature life—forty to fifty years.
In males and females equally.	Five times as frequent in females as in males.
Enlargement of the bones characteristic.	No enlargement of the bones.
Marked prognathism of jaw and flattening of cheeks.	Face full-moon-shaped.
Skin brownish-yellow; hair coarse and unwieldy; nails short and striated.	Skin pale, waxy, shiny, and boggy; hair falls out; nails not affected.
Fingers symmetric and sausage-shaped.	Fingers clubbed at the end.
Administration of thyroid extract is of the smallest benefit.	Thyroid treatment of the greatest benefit.

The **prognosis** is hopeless for cure and doubtful for duration. The disease is progressive, although it remains stationary for a longer or shorter period. Retrogression never occurs. Ordinarily, the patients die of some intercurrent condition; although death may be due to the cachexia of acromegaly itself. Life, however, may last for twenty years after the appearance of the first symptoms.

**Treatment** of the condition itself has proved unavailing. Certain cases have been reported in which there was slight temporary improvement after the use of extract of pituitary body or of thyroid gland, but the results are contradictory. The cephalalgia can be more or less completely controlled by antipyrin or caffeine. Phosphorus, mercury, the iodids, and arsenic have been wholly useless.

## ASTASIA-ABASIA.

ASTASIA-ABASIA is rather a symptom than a disease *sui generis*. It consists of an interference with the power of walking, although the limbs show no trace of paralysis and are capable of performing perfectly other complicated movements. In this respect it is somewhat allied to the functional neuroses. In a case reported by Burr, complicated with severe anemia, changes were found in the posterior and lateral columns of the cord; ordinarily, however, no gross lesions can be discovered. The dis-



ease usually attacks either sex in early adult life, and is sometimes associated with hysteria and neurasthenia.

The **symptoms** consist either of a *tendency to fall* when standing upright or attempting to walk, or of great *difficulty in locomotion*, the feet being dragged along the ground for short steps, the body swaying or making various contortions to maintain the balance, whilst the patient grasps eagerly at any possible support and exhibits every manifestation of fear. Blocq recognizes three degrees. In the (*a*) first or most severe, the upright posture and, above all, walking are absolutely impossible. If the patient is lifted up and supported on either side, the legs hang powerless. These patients, however, when lying in bed show neither loss of power nor incoördination, and are perfectly able to crawl on their hands and knees. In the (*b*) second degree the patient is able to stand if supported on either side; if an attempt to walk is made, the feet are dragged with difficulty along the ground. In the (*c*) third degree locomotion is possible, but the feet are dragged along for short steps, often exhibiting deviations such as occur in ataxia; moreover, the patient is usually unable to proceed as soon as the eyes are closed. In this degree the patients are able to remain standing, but from time to time they exhibit sudden giving way of the legs, followed by equally sudden recoveries. Hysteric stigmata are usually present, although in some cases they fail completely; in two of the cases that I have observed there was complete cutaneous anesthesia.

The **diagnosis** is usually easy, and, though certain cases resemble the movements of the more violent forms of chorea, they may be distinguished by the fact that their limbs become quiet as soon as they lie down. In certain early forms of *locomotor ataxia* also this symptom may be present, and can then only be distinguished from the true form by the lack of coördination in bed and the presence of other tabetic symptoms.

The **prognosis** is favorable, although some cases are quite obstinate.

The **treatment** is chiefly suggestive. Cases complicated by neurasthenia are often cured by the rest-treatment; those in whom anemia is present should be treated for that condition. Hypnotism also may be of use, but the most important element is to encourage the patient to make an effort to walk; it is astonishing then to note the rapidity with which he will ordinarily improve.

## CAISSON DISEASE.

(*Diver's Paralysis; Paralysis from Lessened Atmospheric Pressure.*)

**Definition.**—A paralytic condition caused by sudden transference from an abnormal atmospheric pressure to one of normal intensity.

The **pathology** of the disease is obscure. Leyden has found tears in the substance of the dorsal region of the spinal cord filled with white blood-cells, but without hemorrhagic foci. Other authors have found minute hemorrhages in the substance of the cord and the meninges. It has been supposed by some that as a result of sudden reduction in pressure the nitrogen gases that have been forced into solution in the blood

are suddenly liberated, with the formation of air-emboli; others have believed that the changes are due to a sudden disturbance of the gaseous metabolism. In cases in which death has occurred after a considerable interval the lesions of disseminated focal myelitis have been discovered.

The **etiology** of the disease is very clear, and certain predisposing factors are worthy of note. Divers are more apt to suffer if they have been working at extreme depths, particularly if the period of exposure to great pressure has been prolonged; even moderate pressure will sometimes produce symptoms if continued for a sufficient length of time, and short periods of rest do not prevent the development of the disease. Ordinarily, it can be said that unless the pressure exceeds two and one half or three atmospheres no danger may be apprehended.

The **symptoms** vary greatly in intensity. In the mildest form they consist of *neuralgic pains in the joints*, sometimes with slight articular swelling, *headache*, *giddiness*, and a little *tinnitus*. These pains may become more violent, particularly in the loins, and be followed by a gradual *loss of power* and by *anesthesia* in the limbs; these symptoms may disappear in a few hours or become more severe, with the development of *complete paralysis* and interference with the action of the sphincters. This paralysis usually assumes the form of *paraplegia*; *monoplegia* and *hemiplegia* also occur, and sometimes there are complete paralysis and anesthesia of all four extremities and of the trunk. In the most severe cases *cerebral symptoms* are also present, consisting of sudden loss of consciousness, profound coma, irregular respiratory action, and finally, after a short time, death from cardiac failure.

The **diagnosis** is very easy. It is possible, however, that an attack of *apoplexy* should occur in a man who has been under water, and the patient should always be examined for the presence of this or some other organic lesion.

The **prognosis** varies with the intensity of the symptoms. The lighter forms consist merely of joint-pains and slight dizziness that usually pass away in the course of a few hours. Paraplegias or hemiplegias, developing slowly and not assuming a severe form, are also transient in character. A more severe paraplegia is usually permanent, although some improvement may be expected. The apoplectic forms are almost invariably fatal in the course of a few hours.

The **treatment** consists, firstly, of prophylactic measures. In all places where caisson-work is carried on one or more locks should be provided in which the pressure can be gradually reduced until it is approximately that of the atmosphere. Divers should be instructed to come slowly to the surface. If the pressure exceeds three atmospheres, the maximum length of the working-period should not be more than one hour, and several hours should be permitted between the descents. A chamber should also be provided in which a man who exhibits symptoms of the disease can be once more subjected to a pressure greater than that of the atmosphere, as this usually causes an arrest of the process. When, however, the condition resembles that of acute myelitis, the treatment is purely symptomatic. It consists of rest, careful hygiene, and a stimulating diet. Potassium iodid may also be administered in the later stages, but its value is very doubtful. In some of the acute forms with more or less respiratory failure inhalations of oxygen have been recommended.

## V. VASOMOTOR AND TROPHIC DISORDERS.

## ANGIONEUROTIC EDEMA.

(*Acute Circumscribed Edema of the Skin ; Intermittent Angioneurotic Edema.*)

**Definition.**—A disease characterized by the appearance of an edematous swelling of the skin or mucous membranes. In general it is not accompanied by constitutional symptoms.

The **pathology** of the disease is obscure. It is supposed to be due either to venous stasis or to some nervous influence upon the lymph-channels, causing them to exude liquid. No lesions have as yet been described.

**Etiology.**—Neuropathic heredity appears to have some influence upon the disease, but nervous manifestations in the patient himself are more important. Occasionally the condition follows infectious diseases or severe hemorrhage. The most important exciting causes are cold and emotional disturbances. The disease occurs most frequently in males, and almost exclusively in early adult life.

**Symptoms.**—The *edema* usually appears suddenly, is sharply circumscribed, and the skin of the affected area is slightly elevated and reddened, or else somewhat paler than the surrounding tissue. Ordinarily, subjective symptoms are absent; occasionally there are slight *paresthesie*. The edema may appear in any part of the body, but usually it is most common on the backs of the hands or legs and in the face, especially the eyelid. Occasionally it may appear upon the mucous membranes either of the lips, tongue, or glottis; in the latter situation it sometimes produces severe dyspnea, and at least in one case it has caused death. Its presence has also been suspected in the mucous membrane of the gastro-intestinal tract. Ordinarily the patient has no symptoms whatever of disease; occasionally, however, there are severe colic and sometimes vomiting. In one case hematuria was observed, and in another hemorrhage from the swollen gums; of course in the latter case the diagnosis was doubtful. The patient may exhibit a certain degree of anxiety during the attack. Ordinarily the swelling persists a few days, and then disappears, but relapses are exceedingly common, and may recur very frequently for many years.

The **differential diagnosis** has to be made from urticaria, to which it bears a great similarity. According to Osler, giant urticaria is the same disease.

The **prognosis** is of course favorable for life; for cure it is more doubtful, as the disease is sometimes exceedingly obstinate.

The **treatment** consists of rest, the use of tonics particularly directed to the nervous system, and the correction of any gastro-intestinal disorder. Quinin has occasionally proved very valuable. Hypnotism has also been suggested.



## HYDROPS ARTICULORUM INTERMITTENS.

**Definition.**—A condition characterized by periodic effusions into one or more of the large joints, and usually the knee.

The **pathology** is unknown, but it is suspected that it depends upon some nervous disturbances of the vessels or lymph-channels in the joints.

The **etiology** is also doubtful. The disease occurs in nervous individuals, and has been found associated with other nervous diseases, as exophthalmic goiter, or in patients suffering from other vascular diseases, as angina pectoris.

The **symptoms** consist of the sudden development of a swelling in the affected joint, which, however, does not present any symptoms of inflammation and is rarely painful. This swelling lasts from three to eight days and then disappears as suddenly as it came. At regular intervals of from one to four weeks it is repeated, and this repetition may continue for years.

The **diagnosis** must be made upon the symptoms and the periodicity of the condition.

The **prognosis** is doubtful, most cases being exceedingly obstinate.

**Treatment.**—Electro-therapy in various forms has been recommended. Among the drugs that have been suggested are salicylic acid, quinin, arsenic, and ergotin. None, however, have proved particularly valuable.

## RAYNAUD'S DISEASE.

(*Symmetric Gangrene.*)

**Definition.**—A condition apparently of vasomotor nature, affecting symmetric parts of the body, and chiefly the tips of the extremities.

The **pathology** is by no means definitely made out. It is supposed that alterations must occur in the vasomotor centers of the medulla and cord, but none have as yet been found, partly because the disease is rarely fatal. In some cases peripheral neuritis has been observed and in others peripheral endarteritis. The gangrene is usually superficial, and resembles closely that caused by cold; rarely it causes an extended loss of substance.

The **etiology** of the condition is obscure and complex, largely, no doubt, because a number of different conditions have been confounded under this designation. The disease occurs in children and in neurotic women, less often in men. A neuropathic heredity seems to predispose to it, and occasionally it exists in connection with other nervous diseases, as epilepsy, migrain, hysteria, and mental disorders. The occurrence of paroxysmal hemoglobinuria has led to the suspicion that malaria is an etiologic factor. I am not aware, however, that plasmodia have been found in any case, and the asserted good results following the administration of quinin are insufficient to establish the contention. Syphilis and various other infectious diseases have also been mentioned as etiologic factors, and more recently a form has been described that is sup-

posed to be purely hysteric in nature. The most important exciting cause is exposure to cold, although attacks may also be brought on by severe emotional disturbances.

**Symptoms.**—The disease presents three grades of severity: first, anemia or local syncope; second, cyanosis or local asphyxia; and third, gangrene. *Local syncope* consists in a vasomotor spasm in one or more extremities, the fingers being most frequently affected, and rarely more than one at a time. They become white, almost waxy in appearance, cold, and hard to the touch, and they may be either dry or covered with a cold perspiration. The finger is perfectly numb, but severe neuralgic pains may be felt in the arm; if the skin be pricked with a pin, no blood flows. Ordinarily this syncope disappears gradually, the reaction being accompanied by tingling and formication in the affected digit, which ultimately returns to a normal condition. *Local asphyxia* is a further stage of this condition: in this the finger is blue and swollen, and there is a sense of discomfort that is apparently due to the stretching produced by the engorged veins. This cyanotic condition may also affect the ears, toes, and the tip of the nose, and, like the preceding stage, it may disappear without leaving any trace of its existence. Patients that have reached this stage seem to be more liable to a recurrence upon slight exposure than those who only present local syncope. The attacks are more likely to recur constantly in the same digit, and not to appear first in one and then in another. During the existence of this stage a not infrequent associated symptom is *hemoglobinuria*; this is especially apt to occur in children, and has led to the suspicion of malarial influence. In some cases, when hemoglobinuria is not found, the urine contains an excess of urates. If the attack lasts for several days, *trophic changes* take place in the finger-nail, giving rise to a transverse ridge, which persists until that portion of the nail has grown beyond the end of the finger. If local cyanosis, however, continues sufficiently long, *gangrenous* changes take place. These appear first as small black spots or vesicles filled with serum upon the end of the fingers or about the root of the nail; these gradually slough off, leaving a small ulcer that may slowly cicatrize. Often patients subject to recurrences of the disease show a number of cicatrices on the ends of the fingers, or if the ears are affected there may be slight shrivelling of their edges. The gangrene, however, may be more severe, in which case the distal phalanges of the affected fingers may become black or dark red, covered with blebs, and finally mummified. The line of demarkation then forms, and ultimately the gangrenous portion falls off, leaving an ulcerated stump that slowly cicatrizes. This form may not be limited exclusively to the hands and feet or ears, but symmetric patches sometimes appear in the skin of the breast. During the time that the gangrene is present the patients suffer from excruciating *pains* in the limbs that interfere with sleep, often causing transient melancholia, and seeming, more than the gangrene itself, to depress the general condition. Fever is rarely present; sugar is sometimes found in the urine, but not constantly.

The **diagnosis** is necessarily difficult when it is remembered that all clinicians do not agree that such a disease exists. According to Raynaud's definition, it is a neurosis characterized by enormous exaggeration of the excito-motor energy of the gray parts of the spinal cord that control the

vasomotor innervation. If this be accepted, then all cases in which lesions of the nerves and arteries have been found are not properly of the same nature; and there are a number of other conditions that may produce gangrene, often somewhat symmetric in type and perhaps due to vasomotor spasms (particularly *syringomyelia* and *leprosy*). The so-called "dead finger" is a common symptom, and its occurrence is by no means sufficient justification for a diagnosis of Raynaud's disease. In fact, the typical forms of this condition should advance to slight superficial and symmetrically placed patches of gangrene. Gangrene may also occur in *diabetes*, and hence the urine should always be examined to exclude this condition. *Hysteric gangrene* is rarely symmetric, and the patients present various hysteric stigmata.

The **prognosis** is very favorable. Only in marasmic children do the attacks ever lead to death. Ordinarily they become in time less frequent and ultimately disappear, but in a few cases the tendency to recurrence is obstinate.

The **treatment** consists of improvement in the general condition during the intervals. During the attack the most effective measures are a mild massage, the use of local lukewarm baths, and electricity very cautiously applied, either in a constant descending stream to the spinal column or by the application of the anode to the spine and of the cathode to a vessel containing water into which the hand has been plunged. Amyl nitrite, which might be expected to relax the vasomotor spasm, fails to have any effect; on the other hand, pilocarpin has been employed with good results. If the pains are very severe, they must be combated by morphin—although gangrene may occur at the site of the injection—administered hypodermically, if necessary. Sleep should be obtained by means of narcotics. The gangrenous parts should always be carefully protected by a local dressing.

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## PROGRESSIVE HEMIATROPHY OF THE FACE.

(*Progressive Facial Atrophy.*)

**Definition.**—A rare disease, characterized, as its name would indicate, by a progressive atrophy of one-half of the face, stopping sharply at the middle line, and in the severer forms involving the skin, muscles, and bones.

The **pathology** of the condition is unknown. Rarely symptoms indicating inflammation of the cervical sympathetic, such as dilatation of the pupil or flushing, have been present, and symptoms indicating inflammation of the trigeminus have been equally infrequent. Mendel, however, has reported a case in which he found chronic interstitial neuritis of the branches of the trifacial, and other cases have been reported in which the Gasserian ganglion was diseased. Microscopic examination has shown a disappearance of the subcutaneous fatty tissue and a general atrophy of the elements of the skin itself, often associated with the pres-



ence of an abnormal quantity of pigment. As a rule, the vessels are relatively enlarged.

The **etiology** is unknown. The condition usually commences early in life and shows no predilection for either sex. An hereditary tendency does not appear to exist, but the disease occurs frequently as a complication of, or rather in connection with, other neurotic conditions. Of these the most frequent are neuralgia, migraine, epilepsy, and mental disorders; less frequently, tic convulsif and chorea, particularly if the latter affects the muscles of the jaw and tongue. Occasionally it has been recorded as occurring in patients suffering from locomotor ataxia or multiple sclerosis. It does not appear, however, that progressive facial atrophy has any anatomic connection with these conditions. In a few cases the disease has been preceded by an injury to the skull or face, and in others it has followed an acute infectious disease. Ordinarily it occurs in early life—*i. e.* between the tenth and fifteenth years—and in these cases it usually progresses to the most severe type.

The earliest **symptom** is a flattening of the skin on the affected side, constituting the lightest form of the disease, which may remain stationary at this point; if, however, it progresses, the muscles and bones also become involved, so that the affected half of the face is distinctly smaller than the healthy side. The objective changes that take place in the skin are the development of *white spots* in which the pigment has disappeared, and which have the appearance almost of scar-tissue, or, what is more commonly the case, of an increase in *pigmentation* with a formation of yellowish or brownish blotches, the skin being depressed in these areas, which usually lie along the course of the nerve-trunks, especially the infraorbital. The *hair* becomes thinner, dryer, and often falls out. The secretion of the sebaceous glands is diminished and the skin dryer. Rarer phenomena are the *disturbance of blushing*, so that the affected side of the face remains unchanged in color when, as a result of some emotional disturbance, the other is distinctly reddened. Disturbances of sensation are not common. In some cases electric and tactile sensibility have been diminished; in others the patients have complained of slight paresthesiæ. The special senses remain unaffected, and even when the atrophy extends to the tongue, taste remains perfect on the affected side. In one case there were a slight disturbance of hearing and occasional tinnitus.

The **diagnosis** of the condition is easy both when it is suspected and when it is far advanced. The only condition with which it could be confounded is congenital facial asymmetry. In facial hemiatrophy, however, the skin is shrunken and wrinkled, and the hair is dryer and thinner, contrasting markedly with the healthy side, and there is usually a history of commencement some years after birth. In congenital asymmetry the difference between the two sides is slight, and the skin over the smaller side is normal in every respect. Commonly in this condition we also find differences in the development of the extremities. In a case that I recently observed with marked facial asymmetry, the left side being smaller, the hand and foot on the same side were distinctly smaller than the corresponding members.

The **prognosis** is unfavorable as regards cure. The disease itself is not in the least dangerous, and cases have been recorded that have been under observation for thirty years or more.

**Treatment** is unsatisfactory. The prolonged use of electricity has been said to arrest the process, and sometimes this arrest occurs spontaneously; it is not certain that the treatment is of any use.

An allied condition is **hemihypertrophy of the face**. This is an exceedingly rare condition, and is apparently always congenital. It involves chiefly the soft parts, the ear, skin, tongue, and tonsils being all enlarged. There is an increased secretion from the sebaceous glands, which may appear as small elevations upon the skin. Usually, as in congenital asymmetry, there is enlargement of the extremities on the same side. The only case that has come to autopsy presented no lesions.

*Treatment* is of course unavailing.

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## SCLERODERMA DIFFUSUM.

**Definition.**—A peculiar hardening of the skin, with areas of pigmentation and depigmentation, associated in the more advanced stages with trophic lesions, muscular atrophies, and affections of the bones.

**Pathology.**—The affected skin is characterized by an increase of the connective tissue and of the elastic fibers, and by a narrowing of the vessels as a result of perivascular infiltration.

The **etiology** is not clear. Some of the cases are associated with joint-affections that resemble those of chronic rheumatism; others follow exposure to a very low temperature. The presence of trophic lesions in the skin and the development of myopathies lead to the supposition that it is properly classed with the trophic neuroses. The disease usually occurs in middle life, although cases have been observed among children. Women are more frequently affected than men.

**Symptoms.**—Three stages are recognized: *First*, a rather dense edema. *Second*, a true sclerosis, in which the skin appears thicker, with an absence of the normal folds; it becomes firm and hard, so that it cannot be pinched between the fingers and lifted from the flesh. Moreover, there are always *pigmentary changes*, certain parts being darker than normal, while others become a dead white, appearing almost as if composed of alabaster. The disease, as a rule, attacks first the upper portion of the body—*i. e.* the face, neck, hands, and arms, or the surface of the thorax, and is most pronounced in those regions where the bones are subcutaneous. The *diminished elasticity* considerably interferes with the movements of the body. If the neck is affected, it is difficult to turn the head; if the skin over the joints is involved, their normal flexion and extension cannot be perfectly performed. The subjective sensations are those of tension, the patient complaining that the skin has become “too small” for him. If any forcible action is attempted, there is severe pain, accompanied by slight tears in the skin. The skin is paler and cooler than normal, and the slightest exposure to cold causes great discomfort and cyanosis. The secretion of sweat may be normal, but is usually diminished. Tactile sensibility is unimpaired. The *third stage* is that of atrophy; the skin becomes thin as paper; the other symptoms, however, remain as before, except that the secretion of sweat is abolished and

*ulcerations* appear that either heal slowly or not at all. In addition, there are muscular atrophies associated with contractures. Often there is considerable *atrophy of the bones*, or there may be a development of *exostoses* from the periosteum. Occasionally the end-phalanges of the fingers undergo a process of gangrene that is similar, in some respects, to that of Raynaud's disease. *Chronic joint-affections* may also be observed in this stage, particularly of the fingers.

The **course** of the disease is variable. Usually it develops slowly and lasts for many years.

The **diagnosis** is usually easy, though occasionally it has been confused with *Addison's disease* on account of the excessive pigmentation. There is, of course, some resemblance to *Raynaud's disease*, although the condition of the skin itself is very different. In the atrophic stages it may be confounded with *xeroderma pigmentosum*.

The **prognosis** is always doubtful. In the later stages the patients become emaciated, and pass into a cachectic state, in which death may occur. Pulmonary complications may develop. Complete cure may, however, occur, and particularly in cases that have a rapid course.

The **treatment** is unsatisfactory. The unpleasant tension of the skin may be somewhat diminished by ointments and massage; warm water or steam baths may also give considerable relief. The most important thing is to maintain the general condition of the patient by tonics and a change of climate. Sodium salicylate has been recommended, but is probably valueless.

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## MORPHEA.

(*Scleroderma Circumscriptum.*)

THIS disease consists of the development of *small areas of sclerosis* that are distinctly related to the distribution of the nerves. These areas are round or oval, brownish or violet in color, and as they increase in size there develops in their centers more or less sclerosis. In these sclerotic areas there are often punctiform collections of pigment, the hairs fall out, and superficial ulcerations may be present. Occasionally they may go on to atrophy of the skin. There are no constitutional symptoms.

The **diagnosis** is usually easy.

The **prognosis** as regards life is favorable; as regards cure it is doubtful.

The local **treatment** is the same as for the diffuse form of scleroderma.

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## AINHUM.

THIS is a disease characterized by an enlargement of the little toe and the formation of a line of demarkation at its base.

The **pathology** is not known, but it appears from a Röntgen-ray picture that the bones are absorbed. There is some dispute as to whether



it is one of the manifestations of leprosy or not. At any rate, it does not appear that typical *lepra bacilli* have been found.

**Etiology.**—The disease may occur in childhood or early adult life, and is most common in negroes. It occurs almost exclusively in tropical regions—*e. g.* Brazil and Syria.

The **symptoms** of the condition consist in the formation of a *furrow* at the base of the little toe of one of the feet. This grows deeper and deeper until spontaneous amputation has occurred. Rarely the other toes on the same foot become progressively involved. Certain vasomotor *disturbances* may be observed; the foot is usually swollen, bluish-red, and cold; sometimes the other foot may exhibit similar changes without the formation of furrows at the base of the toes. There is some *diminution of sensation* to touch, temperature, and electricity, and ordinarily the patient complains of vague pains in the limbs.

The **diagnosis** is to be made from leprosy, with which, indeed, it may be identical, and congenital amputation: the latter only occasions difficulty when the disease commences in early life.

The **prognosis** is favorable to life, but the disease is usually slowly progressive.

No effective **treatment** has been discovered, but the parts should be protected against injury, and the patients may be given tonics and anodynes as required.

## ERYTHROMELALGIA (*Weir Mitchell*).

(*Paralytic Vaso-motor Neurosis of the Extremities.*)

**Definition.**—A disease characterized by paresthesia, redness of the skin, and by pain, usually in the toes and heels, associated with more or less severe general disturbances.

The **pathology** is unknown, but the disease appears to be due to some disturbance of the vasomotor centers or nerves.

**Etiology.**—The cause seems to be exposure to cold, but a nervous temperament or a previous attack of rheumatism appears to have some predisposing action. Men are more frequently affected, and the disease usually develops in early adult life.

**Symptoms.**—The earliest symptom, as a rule, is the occurrence of severe *pains in the feet*. Objectively, there are swelling and reddening of the skin, and the sensitiveness is so severe that the patient is unable to walk. The general symptoms consist of headache, dizziness, palpitation of the heart, or even fainting. The attacks occur more frequently during the summer months, and are always aggravated by exposure to heat or a vertical position of the limbs.

The **diagnosis** is easy, the condition being really a symptom rather than a disease. It may occur in the course of hemiplegia and in some organic diseases of the spine, and these should be excluded.

The **prognosis** is favorable; often, however, the disease will recur at irregular periods for a number of years. The attack can usually be cut short by plunging the limb into ice-cold water.

**Treatment.**—This should always be tonic, and employed during the intervals; massage, hot and cold douches, and the faradic current may be used upon the affected extremities. The pain may call for anodynes.

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### ACROPARESTHESIA.

(*Spastic Vasomotor Neurosis of the Extremities.*)

**Definition.**—A disease characterized by abnormal sensations in the hands, slight vasomotor disturbances, and slight stiffness of the fingers.

The **pathology** and **etiology** are not understood. Possibly the condition is due to some disturbance of the peripheral nervous system. It occasionally occurs after injury or as a result of prolonged exposure to cold, hence is common among laundresses. It is more frequent among women than men, and usually develops in middle life.

The **symptoms** consist in the more or less sudden development of *formication* and *tingling* or *numbness* in the fingers and finger-tips, usually bilateral, but sometimes occurring only on one side. Less frequently the toes are affected. These pains are more severe in the night and early morning, and worse in summer or after exposure to heat. The vasomotor disturbances are variable. Sometimes nothing can be observed, and sometimes the extremities are bluish and cold, sometimes pink and warm. Sensibility is rarely affected. In some cases, however, there is considerable hyperesthesia; in others moderate anesthesia. In a few cases there is stiffness of the hands. Slight *trophic disturbances* have been reported in a few cases. The attacks may last from a few minutes to several hours, and may recur frequently or only at considerable intervals. Usually during the attack the abnormal sensations are continuous, but occasionally they are intermittent in character. The condition known as *tender toes*, that occasionally occurs after an attack of typhoid fever, is probably a form of this disease. It is ascribed to the Brand treatment, but incorrectly.

The **diagnosis** is usually easy. Care should be taken, however, not to confuse these acroparesthesiæ with commencing locomotor ataxia, tetany, or hysteria. In Raynaud's disease cold increases the intensity of the symptoms.

The **prognosis** is, in general, favorable, the disease usually disappearing after some months; sometimes, however, the condition is obstinate.

The **treatment** is rather unsatisfactory. Laundresses should be advised to adopt some other vocation. Local stimulation with the faradic brush has sometimes been of value, and hydrotherapy may also be employed. At the same time, the patient should be given tonics, particularly if anemia is present. Alkaline washes are almost a specific for the tender toes. Saturated solutions of sodium or lithium carbonate should be employed.

## MERALGIA PARÆSTHETICA.

(Bernhardt's Disturbance of Sensation.)

**Definition.**—A disease characterized by paresthesia and disturbance of sensation on the outer side of the thigh, in the region supplied by the external cutaneous femoral nerve.

**Pathology.**—Nawretsky has examined one case, and found chronic interstitial neuritis. There is reason to believe that this is not always present.

**Etiology.**—This is very various; some of the cases have been preceded by injury, excessive exercise, or infectious disease. Alcoholism, constipation, and pregnancy are also common predisposing causes; cold douches have been blamed in several instances. The exposed situation of the nerve is supposed to render it more liable to this peculiar disturbance.

**Symptoms.**—These are of two varieties: First, the *paresthesiæ*. There may be burning, tingling, or stabbing pains that are severe enough to disable the patient; or there may be only a feeling of cold or numbness. Second, the *sensory disturbances*. These vary from slight hyperesthesia to total anesthesia. The different senses are not always equally involved; pain, temperature, and electro-cutaneous sensibility being usually more profoundly affected than the others. Frequently both thighs are affected. There is often a tender point just inside the anterior superior spine of the ilium.

The **diagnosis** is easy.

The **prognosis** is doubtful. Some of the cases recover rapidly, but the majority become chronic.

**Treatment.**—But little can be done. Locally, the dry brush seems to do good in some cases, and the general health should be improved if possible.





## PART IX.

# DISEASES OF THE MUSCLES.

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### MYOSITIS.

RHEUMATIC myositis and the suppurative form observed in pyemia, and rarely in other acute infectious diseases, have been appropriately described in connection with the diseases to which they are secondary manifestations. There remain to be discussed two rare forms of the disorder.

#### INFECTIOUS MYOSITIS.

(*Acute Polymyositis*).

**Definition.**—A primary acute or a subacute inflammation of the voluntary muscles due to an unknown microbic agent.

**Pathology.**—The disease is a true inflammation of all the voluntary muscles, involving chiefly the muscular fibers, and to some extent, also, the interstitial connective tissue. Beginning with marked hyperemia, there next occurs an exudation of leukocytes. The muscles are firm, fragile, and later undergo fatty degeneration. Serous infiltration occurs and there is a slight hyperplasia of the intermuscular connective tissues. Hueppe records a case that showed nothing definite beyond a hyaline degeneration of the muscular fasciculi.

**Etiology.**—We are no less ignorant of the predisposing influences than of the specific exciting agency, though, perhaps, young males are most often the victims of this malady.

**Symptoms.**—As a rule, first the muscles of the extremities, and later of the trunk also, become swollen, firmer than normally, and stiff, rendering locomotion somewhat difficult and painful.

The involved parts may also be tender to the pressing finger, and a slight edema may be noticed that is at first more or less localized, but finally becomes generalized, and extends even to the face. An erythematous eruption then appears, which is irregularly disseminated over the skin-surface, and may tend to more or less pigmentation. Moderate pyrexia and splenic enlargement are among the early and constant symptoms. In the advanced stage the muscles of deglutition and of respiration become involved, rendering the act of swallowing difficult, and inducing marked dyspnea.

Among the *complications* may be enumerated bronchitis and bronchopneumonia, the latter often being a terminal condition.

**Diagnosis.**—Taken in the aggregate, the symptoms are of little diagnostic importance and the previous history is invariably negative.

*Trichiniasis* must be discriminated, since this disease produces an identical clinical picture. The distinction may rest upon the examination of an excised piece of affected muscle, which will not only discover the trichinae, if present, but also enable the microscopist to detect the positive evidences of polymyositis. In a recent supposititious case of infectious myositis of my own, a portion of muscle, examined for me by Dr. Babcock, showed neither trichinae nor the histioid changes of myositis. *Multiple neuritis* presents neither swelling nor edema.

**Course and Prognosis.**—The course of the disease may either be comparatively rapid (two or three months), or it may be slow (chronic) and continue over two or three years. It usually terminates in death, which is caused, in the immense majority of cases, by paralysis of respiration. Occasionally, since the heart-muscle has been rarely found to be implicated, the end may be preceded by cardiac failure.

The **treatment** is simply palliative and supportive.

#### PROGRESSIVE OSSIFYING MYOSITIS.

**Definition.**—Myositis, either general or local, in which the affected muscles undergo progressive ossification.

**Pathology.**—Following the changes that ordinarily characterize myositis (swelling, leukocytic exudation, etc.), a calcification that is often complete takes place. The process may extend to and involve the heart.

The **etiology** is obscure, though males are especially the subjects of the complaint, which usually begins about the time of puberty.

**Diagnosis.**—The muscles are represented by plates of bony hardness, leading to more or less complete ankylosis of the joints and vertebrae.

The **course** of myositis ossificans is very slow, and **treatment** has afforded only negative results.

#### PROGRESSIVE SPINAL MUSCULAR ATROPHY.

(*Amyotrophia Spinalis Progressiva; Type of Duchenne-Aran.*)

**Definition.**—A disease of the peripheral motor neurons and the muscles they supply, usually beginning in the cervical region.

**Pathology.**—There is atrophy of the anterior cornua of the cord, affecting chiefly the ganglion-cells, degeneration of the nerve-fibers and of the muscles. Occasionally there are small areas of sclerosis that may involve the pyramidal columns for a short distance.

**Etiology.**—The disease appears to be hereditary in a few cases, and in these may develop in childhood. A commonly accepted predisposing cause is prolonged severe muscular exertion. It is most common in males, and most frequently appears during the third decade of life.

**Symptomatology.**—The first changes usually appear in the *thenar* and *hypothenar eminences* of the hands. These become flat and soft; there are loss of power, some stiffness, and inability to perform delicate coördinated movements; the thumb assumes a position parallel to the other fingers (*ape-hand*); the interossei muscles waste and grooves appear between the metacarpal bones. The degenerative changes do



not ascend by continuity; the deltoid usually being affected immediately after the muscles of the hand. If the two hands have not been affected simultaneously, the other now begins to show characteristic changes. In the lower limbs the quadriceps femoris is usually the first muscle attacked. The disease gradually involves one group of muscles after another until a large part of the muscular system is affected. All the muscles exhibit the fibrillary twitchings, the reactions of degeneration, and the wasting. Hypertrophy never occurs, and the *paralysis* is nearly always flaccid. The fibrillary twitchings are characteristic, but not pathognomonic. They are not constant, but may be developed by slightly irritating the muscle. There is usually quantitative diminution to the faradic, and qualitative alteration to the galvanic current. The *diplegic reaction* consists of the development of contractures in the opposite arm when the anode is placed in the carotid fossa and the cathode over the spine. It is most common in this disease, but may occur in other conditions. The *reflexes* diminish in proportion to the atrophy of the muscles, and ultimately disappear completely; the patients gradually become almost incapable of voluntary motion; but for a time they learn to overcome their disabilities by the compensatory use of other groups of muscles. In the late stage the diaphragm becomes paralyzed and bulbar symptoms appear; usually the patients die from inspiration-pneumonia. Rare and probably accidental symptoms are disturbances of the pupillary reflexes and increase in the secretion of sweat.

**Differential Diagnosis.**—In *chronic antero-polio-myelitis* groups of muscles are affected without any particular order, and total paralysis is a very early symptom; in *amyotrophic lateral sclerosis* the spastic symptoms are present; in *syringomyelia* and *pachymeningitis cervicalis hypertrophica* disturbance of sensation, pain, and trophic lesions occur; in *Pott's disease* affecting the lower cervical region there are tenderness over the spine and sensory disturbances; in *peripheral neuritis* the fingers are unequally affected and the deltoid does not waste; in *arthritic atrophy* joint-symptoms are present; and in the peculiar *muscle-atrophies* following excessive use of certain groups of muscles, rapid improvement occurs when the cause is removed.

**Prognosis.**—This is unfavorable as to cure. The course is exceedingly slow, and the patients often live for a number of years after the first symptoms have appeared. They are, however, exceedingly liable to pulmonary complications, particularly a fatal form of bronchitis.

**Treatment.**—Prophylactic measures, such as the avoidance of prolonged excessive work, are rarely possible. Retardation may possibly be obtained by the systematic use of electricity, massage, and gymnastics. Gowers advocates the hypodermic injection of strychnin nitrate in ascending doses, commencing with  $\frac{1}{100}$  gr. and rapidly increasing to  $\frac{1}{40}$ ; one injection should be given daily.

## PROGRESSIVE NEURAL MUSCULAR ATROPHY.

(Charcot-Marie-Hoffmann Type; Peroneal Type, Gowers.)

**Definition.**—A degenerative process, apparently commencing in the nerves, and characterized by muscular degeneration, with subse-

quent contractures, marked sensory disturbances, and a loss of the reflexes.

**Pathology.**—Degenerations have been found in the muscles, the peripheral nerves, and the spinal column. In the former the muscle-cells show degenerative changes. The nerves exhibit a chronic interstitial neuritis with proliferation of the connective tissue, and destruction of the myelin-sheaths and axis-cylinders. In the spinal cord degeneration has been found in the posterior columns. The nature of the disease seems to be akin to that of neuritis, the changes in the muscles and spinal cord being secondary to those of the nerves.

**Etiology.**—Heredity seems to play an important part in the causation of the disease, which may either occur in successive generations of a family or affect several members of the same generation. Sporadic cases occasionally occur for which it is impossible to trace any ancestral influence, though, as the disease has been known to skip a generation, it is not impossible that such cases are still hereditary. Males are much more frequently affected than females, and the disease almost invariably commences between the ages of ten and twenty years.

**Symptoms.**—As the name implies, muscular wasting usually begins in the muscles of the feet or hands, either the peronei, the common extensors of the toes, or the small muscles of the foot itself, or else in the muscles of the thenar and hypothenar eminences and the interossei. Usually the atrophy is symmetric. In the feet it leads to an early development of club-foot, which is most pronounced when the extremity is at rest. Very early the atrophy of the small muscles causes the toes to assume the claw position, and the atrophy of the peroneals causes foot-drop, so that in walking the foot is dragged along the ground. In the later stages the foot becomes permanently fixed in a position of equino-varus or valgus. The hands have the characteristic appearance given by a flattening of the ball of the thumb and middle finger. The interosseal grooves also become deeper and the fingers gradually assume the claw-like position ("*main en griffe*"). The disease extends slowly upward, involving the muscles of the calf and thigh or the forearm and arm. The affected muscles usually show distinct fibrillary twitchings that may be so severe as to give rise to an irregular tremor of the fingers. Spontaneous spasmodic contractions may also occur. When electrically examined the muscles either show a marked diminution in reaction to the galvanic and faradic currents, or distinct reactions of degeneration can be elicited. Similar electric changes are also found in the nerves. Mechanic excitability of the muscles is considerably diminished, these changes being found also in the muscles that are apparently healthy. The tendon-reflexes are usually absent, although in the early stages, when the muscles of the thigh are still unaltered, the knee-jerk may be merely diminished. Sensation is sometimes unaltered, but ordinarily there is considerable diminution to touch. It is possible that some cases show an alteration in the pain and temperature sense; often there are paresthesiæ, and occasionally, pains of considerable intensity. The general condition of the patient, however, remains excellent. The vegetative organs are unaffected and nutrition is therefore intact.

The **diagnosis** can be made from other forms of progressive muscu-

lar atrophy (particularly the type "Duchenne-Aran") by the sensory disturbances; from *locomotor ataxia* by the absence of sphincter disturbances; and from the *cerebral palsies of childhood* by the fact that it begins late in life and is distinctly progressive, showing also a diminution of the tendon-reflexes and reactions of degeneration in the muscles.

The **prognosis** is good as regards life, but unfavorable as regards cure or even improvement. The course of the disease is extremely slow.

The **treatment** employed in the other forms of amyotrophy may be tried, but so far nothing has succeeded in staying the course of the disease.

A type of disease closely allied to the preceding has been described by Déjerine under the name of "*infantile hypertrophic and progressive interstitial neuritis*." The muscular symptoms were the same, but there were in addition ataxia, lancinating pains in the limbs, considerable sensory disturbances, Romberg's sign, myosis, with slow or absent pupillary reflexes and nystagmus. In addition to these a peculiar symptom in his case was the enormous hypertrophy of the nerve-trunks, which could be felt under the skin as large, firm cords. Pathologically the muscles showed degenerative changes and the nerves a pseudo-hypertrophy due to the enormous proliferation of the connective tissue and degeneration in the posterior columns of the spinal cord. The disease appears also to be due to old hereditary influence, the first 2 cases described being a brother and sister.

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## PSEUDO-HYPERTROPHIC MUSCULAR PARALYSIS.

**Definition.**—A disease characterized by a progressive loss of power in the muscles without disturbance of their electric reaction, while at the same time one or more of them increases in size and firmness.

**Pathology.**—Microscopic examination of the muscles shows that the hypertrophy has been produced by the hyperplasia of adipose tissue in the perimysium internum. The muscle-fibers may either be normal, atrophied, or hypertrophied, and there may be a relative increase in the connective tissue. The motor nerves are invariably intact.

The **etiology** of the disease is obscure. It appears to be transmitted by females chiefly to males. Occasionally it has been associated with mental disturbances that appear to indicate that it may be indirectly a nervous condition. Consanguinity, according to Gowers, may be a predisposing cause if it continues through several generations. The disease usually develops in early life, and those forms that occur after puberty are more common in females.

**Symptoms.**—The enlargement usually affects the muscles of the calves of the legs, although various muscles in other parts of the body may be involved, as the infraspinatus and masseter, or the muscles of the arms and thighs, giving the patient the appearance of an unequally developed athlete. Fibrillary contractions may sometimes be seen, but are not frequent. The electric reactions show no qualitative alteration, but are quantitatively diminished in proportion to the loss of power. This



loss of power is manifested first in the gait, which is uncertain and waddling; next, by the difficulty the patient has in arising from the ground. He first gets on his hands and knees, then lifts his knees from the floor and, placing his hands first on his ankles, climbs up his legs until he assumes a more or less upright position (Fig. 78). In

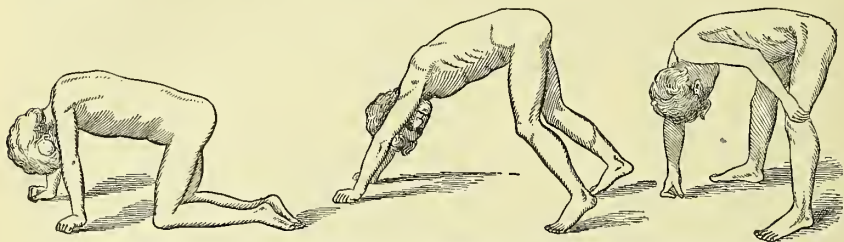


FIG. 78.—Mode of rising from the ground in pseudo-hypertrophic paralysis (Gowers).

the later stages of the disease the volume of the muscles becomes less than normal. At this period contractures may occur leading to the development of club-foot or of lateral deviation of the spine. Lordosis may also be produced by weakness of the muscles of the back, and the spinal column, being no longer properly supported, may topple to one side or the other. Ultimately the patient may lose all power in the affected limbs and pass into a cachectic state, in which he dies. Few ever reach adult life. Some of the cases, however, seem to be milder in character, and may amount to nothing more than a slight weakness, which persists throughout life but does not seriously inconvenience the patient. Often signs of intellectual disturbance are present, the patient learning more slowly and showing an impaired intellectual coördination. At other times epilepsy may be present. A peculiar variety is known by the French as *forme fruste*; this is characterized by a rapid atrophy of the hypertrophied muscles, and consequently the course of the disease is more severe.

The **diagnosis** is relatively simple, a typical case being easily recognized. In those cases, however, in which hypertrophy is slight, the disease may be easily confounded with progressive muscular dystrophy, a disease of which, perhaps, this is only a variety.

The **prognosis** is unfavorable, few of the cases living to adult life.

**Treatment.**—No tonic or alterative drug has exhibited the power of arresting the progress of the disease, and electricity is equally valueless. Gowers believes that persistent, systematic exercise and massage sometimes retards the course, but never leads to arrest. This should be tried in every case.

## DYSTROPHIA MUSCULORUM PROGRESSIVA (*Erb*).

(*Scapulo-humeral Type*; *Juvenile Form of Progressive Muscular Atrophy*.)

**Definition.**—A primary myopathy, commencing usually in the muscles of the shoulder-blades and appearing about the period of puberty.

**Pathology.**—There are irregular hypertrophy and atrophy of the

muscle-fibers, disturbances of the striation, and multiplication of the nuclei, with a relative increase of the entire fascicular connective tissue. The motor nerves, even in their finest terminations in the muscles, show no alteration, nor are changes found in the cord.

**Etiology.**—Hereditry plays an important part, although occasionally the disease may occur sporadically. As in the other forms, it sometimes develops from emotional disturbances, exposure or fatigue. The sexes are affected about equally. The first symptoms usually appear at puberty, or not later than the twentieth year, although in a few cases the condition has developed in early adult life.

**Symptoms.**—The muscles first affected are usually the pectorals and the latissimus dorsi. From these the process rapidly extends to the muscles in the neighborhood—*i. e.* the serrati and the muscles of the back. The muscles of the arm, particularly the flexors in the lower arm and the long extensors, are usually most involved. In the thighs, the glutei and quadriceps femoris are particularly subject to the atrophic process. The muscles that are most likely to escape are the sterno-mastoid, the spinati, and the deltoid in the upper part of the body; and the sartorius and the muscles of the calves of the legs in the lower part. The muscles gradually waste, and the wasting is accompanied by a corresponding loss of power, a diminution in the reflexes and of the electric reactions. Reactions of degeneration are not present. Certain peculiar appearances are produced by the atrophy of certain of the groups of muscles. As the shoulder-blades are no longer supported, they stand out from the back, giving rise to the so-called “winged” appearance, and as the result of the weakness of the muscles of the back lordosis is exceedingly common. Finally, weakness of the muscles of the back, and particularly of the glutei, causes the patient, when he rises from the stooping posture, to go through the same actions that are carried out by children suffering from pseudo-muscular hypertrophy—*i. e.* climbing up his own legs. Finally, if the diaphragm is involved, dyspnea may develop to a greater or less extent, and may even lead to death. In certain rare cases the muscles of the face also show slight paresis, manifested by an inability to whistle, a disturbance of speech, and an imperfect closure of the eyelid. Motion is affected proportionately with the degree of atrophy. The gait is disturbed and becomes waddling, due to the alternate lifting of the sides of the pelvis in order to clear the foot of the ground. Sensation is never disturbed. The sphincters are not involved and bulbar symptoms do not appear, even late in the disease.

The **diagnosis** must of course be made from the other forms of progressive muscular atrophy. The differential diagnosis is essentially the same as that for the *infantile type*, excepting that from the latter it can be distinguished by the different order of invasion and the period of life at which it occurs. It is exceedingly difficult, sometimes, to make a differential diagnosis if the muscles of the face are also involved. In certain rare cases, such as one described by Oppenheim, there may be a congenital absence or a weakness of certain groups of muscles, and particularly of those most likely to be involved in this disease. In such instances the differential diagnosis may be made upon learning that the weakness has existed since birth and has not increased.

The **prognosis** is hopeless as regards improvement. The course of

the disease is slow, but progressive, though life is usually not threatened unless the diaphragm is involved. The patient ordinarily lives for twenty or thirty years after being first attacked.

The **treatment** consists of systematic gymnastics, massage, and electricity. At the same time the general health of the patient must not be neglected. Apparent results are, however, rarely attained.

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### DYSTROPHIA MUSCULORUM PROGRESSIVA (*Déjerine-Landouzy*).

(*Facio-scapulo-humeral Type; Infantile Type of Progressive Muscular Atrophy.*)

**Definition.**—This form is characterized by the development of muscular atrophy of the face, shoulder, and arm, giving rise particularly to the *facies myopathica*.

**Pathology.**—The wasted muscles show in all respects the histologic changes found in the type of Erb, and essentially those found in the pseudo-hypertrophic form of muscular paralysis.

**Etiology.**—The disease is distinctly hereditary, and occurs ordinarily about the third and fourth years with equal frequency in both sexes, although a curious predisposition to one sex or the other is noted in certain families. As many as ten generations in a family have been recorded in which one form or other of myopathy developed. In a few cases some acute infectious disease or disturbance of general nutrition has preceded the muscular wasting, but there is no reason to believe that the connection is other than accidental.

**Symptoms.**—The disease usually begins in the muscles of the face. Of these the muscles of the eyelids and mouth first undergo degeneration, giving rise to a peculiar expression in which the eyes cannot be closed, the upper lid covering only half of the eyeball; the under lip drops forward and downward; the upper lip is wasted and expressionless; all wrinkles disappear, and the patient has a peculiar and strikingly stupid expression. The ordinary movements of the face are considerably affected. Whistling cannot be accomplished, speech is imperfect, and when the muscles of the eyeballs are involved ocular fixation is impossible. The shoulder-muscles next undergo atrophy. The earliest to be affected are usually the *cucullaris*, the rhomboids, and the pectoral muscles; finally, the disease extends to the arms, where we find the deltoid, biceps, triceps, and some of the extensors involved. Ordinarily certain groups of muscles seem to escape, among these being the muscles of mastication and the muscles of the forearm and hand. As the result of these changes, the shoulder-blades become more freely movable and stand out from the back and the shoulders, and when the patient is lifted by placing the hands under the arms, the shoulders show an abnormal degree of upward movement. The nutrition of the muscles is only affected in proportion to the atrophy. Electric reactions remain normal qualitatively, but are diminished quantitatively. Fibrillary contractions occur with extreme rarity; power is diminished in proportion to the wasting. Sensory disturbances do not occur.



The **diagnosis** is to be made from the *spinal* and *neural forms* of muscular atrophy and from the *congenital absence* of certain groups of muscles. From the two first-mentioned forms it can readily be distinguished by the fact that the hand becomes involved, if at all, in the last stages of the disease; also by the absence of the reactions of degeneration and of muscular twitching. It is also diagnosed from the neural type by the absence of disturbances of sensation. From the congenital absence of certain groups of muscles the diagnosis is sometimes difficult, for, curiously enough, the groups of muscles affected are usually the same as those affected by the myopathy. A distinction can be made partly by the history, partly by the more efficient and perfect compensatory hypertrophy of the muscles that remain.

The **course** of the disease is slowly progressive, only occasionally exhibiting a temporary arrest.

The **duration** is variable, but patients may live thirty or forty years after the first symptoms appear.

The **prognosis** is of course hopeless as regards cure or improvement. As regards existence, however, it is the most favorable of all the forms of progressive muscular atrophy—a fact that is probably due to the ability of the patients to walk until the very last stages of the disease, so that they are able to maintain a better physical condition.

The **treatment** is the same as that for other forms, and consists of electricity, massage, and especially of systematic gymnastics.

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### HEREDITARY MUSCULAR PARALYSIS (*Leyden*).

THIS commences in children, and usually between eight and ten years of age. It affects the muscles very much as they are affected in the pseudo-hypertrophic form, except that there is no increase in size. The disease is markedly hereditary in type.

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### ARTHRITIC MUSCULAR ATROPHY.

**Pathology.**—It has frequently been observed that after inflammation of a joint the muscles that move it have undergone a certain degree of atrophy. This usually occurs in the extensors, and is severe in proportion to the duration of the inflammation. Microscopic examination of the muscles shows a rather uniform diminution in the breadth of the fibers, as well as a slight proliferation of the nuclei and occasionally an indistinctness of the striation. The nerve-trunks and cord have been reported to be normal.

The **etiology** of the condition is not clearly determined. It has been supposed to be due to disuse, but if such were the case all the muscles moving the joint would be equally affected. Moreover, it sometimes occurs too rapidly to render this explanation acceptable. It has also been supposed to be due to the extension of the inflammation either to the nerves or directly to the muscles, but the other symptoms of neuritis are rarely present. Finally, Vulpian has suggested that it is of reflex origin, and this hypothesis is most generally accepted.

**Symptoms.**—The wasting usually occurs very rapidly after the onset of the joint-affection. The muscles show a diminished contractility to faradism and galvanism, but the reactions of degeneration do not occur. Occasionally there is fibrillary twitching. The mechanic irritability of the muscles is greatly increased, and the reflexes show a corresponding exaggeration, ankle-clonus being frequently observed when the knee- or ankle-joints are affected.

The **diagnosis** may be readily made upon the existence of the joint affection, the local character of the muscular atrophy, and the absence of degenerative reactions with increased mechanical irritability.

**Prognosis.**—Ordinarily, as soon as the joint has recovered, improvement commences in the muscles and progresses rapidly to complete restoration of function. In some cases, however, atrophy persists, and in a few instances secondary contractures take place.

The **treatment** consists, first, in the removal of the cause by the cure of the articular condition; secondly, in gentle massage and electric stimulation of the muscles. As a rule this should not be commenced until the joint is well.

#### MUSCULAR ATROPHIES.

These may also occur as a result of other conditions, such as direct injury, fracture of the bones, or prolonged work with a single group of muscles, but they scarcely demand separate description.

#### MUSCULAR HYPERTROPHY.

This occasionally occurs as an idiopathic affection. In these cases microscopic examination shows an increase in the size of the fibers, although sometimes there are slight degenerative alterations, such as the presence of vacuoles or indistinctness of the striation. The cause of the disease is unknown. It occasionally appears in those of a neuropathic heredity, and one case is recorded that developed in an idiot. The symptoms consist of enlargement of the muscles, which usually exhibit increased power, but, at the same time, great susceptibility to fatigue. Occasionally the power is diminished.

The *diagnosis* from pseudo-muscular hypertrophy is sometimes difficult. The *prognosis* is unfavorable for any improvement in the condition. No *treatment* that has any influence upon it is known.

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### THOMSEN'S DISEASE.

(*Myotonia Congenita.*)

**Definition.**—An hereditary disease of the muscles in which the groups that have been contracted by a voluntary influence remain for a short time in a state of contraction, and then relax slowly.

**Pathology.**—Certain authors have described alterations in the terminal nerve-plates in the muscles, but it is difficult to determine whether these alterations are artificial or an actual part of the disease. The peripheral nerves are normal. The muscles themselves exhibit the follow-

ing alterations: The muscle-fibers are, on the average, of an increased transverse diameter—*i. e.* the smallest are the size of ordinary muscle-fibers and the largest about twice the size. There is also a distinct and considerable increase in the number of nuclei. The protoplasm is not so clear as in normal muscles, but shows a fine granular cloudiness, rendering the striation less distinct. Occasionally, the muscle-fibers are vacuolated. The connective tissue between the muscle-fibers is normal.

**Etiology.**—Hereditary influence is the most important factor in the causation of the disease. Thomsen, who was himself a victim, has been able to trace the disease for five generations in his own family. Occasionally a generation is skipped. Other factors that have been supposed to act as predisposing or exciting causes are prolonged exertion (1 case having developed in a man without myotonic antecedents after two years of severe exertion) and emotional disturbance of the mother during pregnancy. Exposure to cold, and fright, and a neurotic temperament have also been accused of exerting a predisposing or exciting influence. The disease is somewhat more frequent in males than in females, usually develops in early life, is often associated with manifestations of mental disturbance, and occasionally occurs in those whose ancestors have exhibited lesions of the nervous system other than myotonia.

**Symptoms.**—The chief symptom of the disease is the so-called myotonic contraction. If the patient, after a period of rest, attempts to set a certain group of muscles in action, the first contraction is made, but is not followed by relaxation for a considerable interval—sometimes as much as a half minute; during this period the muscles remain in a state of tonic contraction. Thus, if the patient attempts to shake hands, he clasps the other hand strongly, and the clasp persists. When he lets go, it is seen that a slight degree of tonic contraction still exists, for it is impossible for him to straighten out his fingers immediately. Upon a repetition of the movement the tonic contraction recurs, but not so strongly, and if the repetition is continued, it disappears entirely, so that the muscular system of the patient behaves in all respects like that of a normal person, and long walks or other severe muscular exertion may be undertaken. In some cases practically the whole muscular system is affected, although, excepting the muscles of mastication, the muscles of the face usually escape. In others the disease is limited perhaps to the upper, perhaps to the lower, extremities. In the former condition the patient may, upon an attempt to make a vigorous motion after resting, suddenly become rigid and fall to the earth with considerable force, often injuring himself severely. He will then lie upon the ground perfectly conscious, but incapable of relaxing his muscles. When the disease, as is more frequently the case, is limited to the lower extremities, the chief disturbances observed are in walking. The first step is accomplished, whereupon the patient halts, both legs having become fixed; after a time they relax and another step is taken. The period of delay is now much shorter, and after a few more steps disappears entirely. The severity of the contraction is diminished by moderate exercise, heat, and tranquillity of the spirits, and is increased by excitement, cold, and fatigue. The muscles of deglutition and the sphincters and the muscles belonging to the non-striated muscular system are never involved. Pain is not present, except perhaps a slight sensation of cramp, nor are there disturbances of sensa-



tion. Mental disturbances are frequent, and have been ascribed to the anxiety occasioned the patient by the disease. They consist of irritability, the avoidance of society, and sometimes of melancholia. The reflexes show various modifications; the knee-jerks may be either normal, increased, diminished, or absent. The most important pathognomonic symptoms are the alterations in the electric reactions of the muscles. The changes are as follows: Mechanic irritation of the motor nerves is normal or diminished; the mechanic irritation of the muscles is increased, and so modified that the contraction instead of being sudden is slow, with a long tonic after-contraction. The faradic irritability of the nerves is normal, and faradic excitation of the muscles produces a tonic contraction of long duration. The galvanic irritability is quantitatively increased and qualitatively altered; that is to say, ACC is equal to and sometimes even greater than KCC. All the contractions are slow, tonic, and of long duration. Finally, the application of the constant galvanic stream gives rise to rhythmic contractions that pass along the body of the muscles in slowly moving waves at the rate of about one to three per second. Occasionally qualitative galvanic alterations have been observed in the nerves. Finally, the appearance of the patient is of some value. The muscles are developed almost as much as those of an athlete, without a corresponding increase of power.

The **diagnosis** is usually easy, and particularly if it be possible to examine the electric reactions. The condition might possibly be confounded with *pseudo-hypertrophic muscular paralysis*, in which the muscles are also considerably developed; but instead of being normal they manifest greatly diminished power and fail to give a myotonic reaction. From *tetany* the condition may be distinguished by the absence of Trousseau's sign, by a briefer period of tonic contracture, and an absence of severe pains. From *spastic paraplegia* and *Little's disease* it may be distinguished by the fact that in these diseases the spastic conditions are permanent and do not disappear after exercise. From *occupation-neuroses* it may be distinguished by the fact that the cramps only appear upon the performance of a certain peculiarly coördinated movement. From *hysteria* it is differentiated by the absence of stigmata and the care an hysterical patient exhibits to avoid injury to himself, and by the peculiar electric reaction.

The **prognosis** is hopeless. The disease commences in early life and continues until death, with more or less frequent remissions and exacerbations. It is possible that these remissions may be permanent, and one case has been reported of a young woman whom marriage greatly benefited. The disease is rarely dangerous to life, excepting in so far that those who suffer from it are much more liable to injury.

**Treatment** is exceedingly unsatisfactory. Practically nothing can be done, although in a few cases systematic stimulation of the muscles has produced some mitigation. The patients often learn methods by which they can at least diminish the unpleasant symptoms. Certain movements seem to prevent or shorten the period of tonic contraction. Of course exposure to cold or emotional disturbance should be avoided as far as possible.

## PART X.

# THE INTOXICATIONS; OBESITY; HEAT-STROKE.

## THE INTOXICATIONS.

### ALCOHOLISM.

(*Alcoholic Inebriety.*)

**Definition.**—An acute or chronic intoxication due to the abuse of alcohol. It is a general degenerative condition, particularly of the brain and nervous system, characterized by a moderate (often progressively increasing) or excessive, continuous or periodic, craving for alcohol, leading to drunkenness. Alcoholism is often simply a variety of *inebriety* or *narcomania*, a congenital or acquired brain- and nervous disease, characterized by a resistless, permanent desire for alcohol (*alcoholic inebriety*). *Mania-a-potu*, or “crazy drunkenness,” is an acute maniacal condition occurring in an alcoholic drinker of a neurotic constitution. *Delirium tremens* is an hallucinatory manifestation that occurs in habitual drinkers of alcohol, either as the direct consequence of the long-continued action of alcohol on the brain, or because of its sudden withdrawal in an inebriate. *Dipsomania* is an alcoholic insanity in which an intense maniacal “drink-impulse” occurs in a periodic drinker (usually of spirits).

**Pathology.**—In cases of death from acute alcoholism the brain and kidneys are found to be greatly engorged with blood. The gastroduodenal mucous membrane is also markedly congested, injected, and covered with a thick, sticky, blood-tinged mucus.

**Chronic Alcoholism.**—Since alcohol is physiologically a poison, and not a food, and essentially a drug, and not a drink, the effects of its habitual ingestion are directly to produce degeneration of nearly all of the bodily tissues, and indirectly to increase the liability to many diseases by lessening the systemic powers of resistance, thus favoring fatality from such disease. The degree of pathologic change depends upon the innate vigor of the tissues, the age at which indulgence in alcohol is commenced, and upon the kind, degree of concentration, and the quantity of alcohol habitually taken. Ethylic alcohol is less deleterious than the “fusel oil” that is sometimes used as an adulterant in spirits.

The chief effects of chronic alcohol-poisoning are seen in the nervous and digestive systems, and in the kidneys. Fatty changes are prominent in the malt-liquor intemperates, while a connective-tissue over-

growth predominates in spirit-drinkers. The mucosa of the stomach presents the appearance of chronic gastric catarrh. Dilatation of the stomach is common in free drinkers of beer, alc, and porter. The liver shows the changes of chronic congestion, of fatty infiltration or degeneration, or of cirrhosis and contraction. The renal changes are analogous to those of the liver, the chronic congested ("pig-backed") and fatty kidneys occurring mostly in those who have drunk excessively of malt liquors, while the small, sclerosed, and fibrous kidneys (chronic interstitial nephritis) are seen in those who have been spirit-habitués. The heart is often loaded with fat, and the muscular structure may reveal fatty degeneration, being pale, flabby, friable, and dilated. The blood-vessels are atheromatous, thickened, tortuous, and sometimes varicose, and sudden death has been caused in inebriates by the rupture of small aneurysms of the middle cerebral artery. In the brain the various stages of sclerosis, with shrunken, narrow, and flattened convolutions, often appear. Chronic pachymeningitis, with slight hemorrhages, is not infrequent. The pia-arachnoid membrane also may be opaque and thickened, and serous effusions into the subarachnoid space and into the ventricles have been noted. The nerve-cells, nerve-centers, and nerve-fibers show degeneration, hardening, and atrophy. Alcoholic neuritis is especially prominent in many cases.

**Etiology.**—An impaired personal health and vigor, as well as the "personal equation" and a deficiency of will-power, self-control, conscience, and conviction, are among the *predisposing causes*. Drunken or inebriate parents frequently transmit to their offspring a morbid impulse or desire for alcohol, and an environment of depraved morality and of depressing and corrupting social influences are usually potent disposing influences, particularly in those who are illy prepared, by heredity or training, to resist the temptation and insidious activities of such evil surroundings. Although some assert that poverty predisposes to intemperance, it is more likely that, in a great majority of instances at least, intemperance is the cause rather than the consequence of poverty, both of individuals and communities. The *exciting cause* is the misuse of alcohol in the form of distilled liquors or spirits (fermented liquors, wines, and malt liquors). "The more concentrated the alcoholic liquor ingested, the more intense the inflammation of the tissue. At the same time, an equal quantity of any of the potable alcohols will sooner exhibit its characteristic symptoms if largely diluted with water."

**Symptoms.**—The symptoms of acute alcoholism range from mild intoxication to an acute delirium or a profound stupor and coma. It begins with the stage of *vascular relaxation* and of feelings of warmth and exhilaration, due to the depressing and paralyzing effects of the alcohol upon the vasomotor tone. The second stage is one of *partial functional paralysis of the nerve-centers*, marked disturbance of the faculties, muscular incoördination, and delirious speech. In the third stage, of "dead-drunkeness," there are acute coma, stertorous breathing, a bloated and congested face, a slow and full, but weak, pulse, a cold and clammy skin, a heavy alcoholic odor of the breath, and, sometimes, incontinence of urine and feces. It frequently happens that unconsciousness is not so profound but that the patient may be aroused, though replies to questioning are stupid and incoherent.



Ordinary acute alcoholism seldom passes beyond a stage of exhilaration, ending in mild narcosis. Sometimes, however, the irritant action of the alcohol predominates over its narcotic action, giving rise to acute alcoholic gastritis or nephritis.

**Acute mental disorders** ("acute alcoholic insanity") are not infrequently met with. *Mania-a-potu* may come on quite suddenly in debauchees, or in those who have drunk hard during a short time, as in a night's carousal. The mental excitability increases until a violent maniacal storm not unlike the mania of epilepsy possesses the drinker. While in this state of infuriated delirium homicide may be committed. Tremors are absent. Acute *alcoholic melancholia* develops suddenly in some cases, with a suicidal tendency. *Delirium tremens* is more common in alcoholic inebriates, and is also seen at times in those who drink greatly to excess, but are not habitués. Convulsive seizures have been noted in some cases, interrupting the coma ("acute alcoholic epilepsy"); these may or may not be accompanied by mania. An acute *alcoholic paralysis* from multiple neuritis (occasionally with ataxic symptoms) may attack hard drinkers, and may last for several weeks or months.

**Chronic alcoholism** (alcoholic inebriety) I consider a true disease. While acute alcoholism may also be an occasional manifestation of the chronic affection, it is often a vice which, if indulged in to an excessive degree, or if too frequently repeated, becomes a disease, though it is difficult to determine at what point the transition occurs. Again, it is not always easy to learn whether the early acute alcoholic excesses are really vices or morbid, diseased cravings for alcohol in hereditary narcomaniacs. The disease of inebriety (alcoholic) is a condition in which, as some one has said, it is not whether one "cannot" or "will not;" but in which one "cannot will" to resist the desire for alcohol.

The steady, so-called "moderate drinker" who saturates his blood and tissues every day for years is much more apt to suffer from chronic alcoholic poisoning with its attendant degenerations than one who goes on a "spree" once a month for a day or two, and during the intervals is free from the toxic influence of alcohol. The *symptoms* develop very gradually, and are usually marked for some time by the deceptive sensation of stimulation, warmth, and well-being, due to the vasomotor paresis and the anesthetic effects of the alcohol. Impairment of digestion is early noted. There are a coated tongue, foul breath, vomiting before breakfast, and gastric distress after eating. Constipation alternating with diarrhea is common. Muscular tremors gradually develop and often progress into an ataxic gait. Insomnia, mental impairment, and blunting of the moral sense come on. "Alcohol dims the perception, confuses the judgment, paralyzes the will, and deadens the conscience" (Kerr). In his distress and degradation the inebriate seeks to relieve himself by taking more of the alcohol, only to find, on awakening from his narcosis, that body, intellect, will, and emotion are still more depraved. In fact, the brain- and nerve-disorders are more grave, permanent, and extensive in the majority of instances than those of the viscera. This is owing to the delicacy of the nervous mechanism and to the ready degeneration under the influence of the altered blood, and the consequent impaired cellular nutrition, directly due to the toxic action and

deficient normal pabulum, and indirectly to the lessened elimination of waste-products.

**Dementia** is often the terminal state of the chronic inebriate. Delusions of persecution are quite masked in alcoholic insanity. The depurative organs manifest various symptoms due to the long-continued irritating action of alcohol. The liver is either fatty and enlarged, or cirrhotic and contracted, and jaundice, dropsy, and hemorrhoids, along with physical hepatic signs, are correspondingly observed. The watery eye, the injected conjunctivæ, the swollen eyelids, the bloated and flabby or pallid and shrunken face, the dilated capillaries of the nose (*acne rosacea*) and cheeks, may now be seen. The urinary examination will show in many cases the deranged function of the kidneys and point to the nature of structural impairment. On account of the weak and flabby heart there are palpitations, dyspnea, and precordial distress, and occasionally sharp pains. Chronic valvular endocarditis may be discovered. The pulse is soft and weak in beginning fatty degeneration of the vessels. Thickened arteries are common in old cases, and the pulsations are often increased in tension and usually rapid. Muscular capacity and endurance are greatly diminished.

**Delirium tremens** occurs in the majority of cases in inebriates or chronic drinkers during or after a debauch, and particularly from the use of spirituous liquors. It may occur, also, during abstinence from alcohol, on account of some mental perturbation, or fright, accidental shock, or acute inflammatory illness. It may either come on suddenly, or be preceded (often for a day) by some slight premonitory symptom, as anorexia, restlessness, or depression of spirits. The patient usually awakens at night with a tremor, becomes sleepless, wants to get out of bed to do some imaginary thing, talks constantly and incoherently, looks about uneasily and fearfully, and breaks gradually into a cool perspiration. Hallucinations of sight, hearing, and smell develop. The patient sees terrifying and loathsome reptiles, and tries to escape from them, or to clutch them in order to cast them away. The "horrors" may become so great that suicide may be attempted, as by falling out of the window. Auditory hallucinations may take the form of enemies, policemen, or the roar of wild animals. The muscular tremors increase, the pulse becomes frequent and weak, and the tongue coated with a thick white fur. There is moderate fever, which, if the delirium is prolonged, takes on a typhoid character, the tongue becoming tremulous, dry, brown, and fissured, with the onset of *subtulus tendinum*, *carphologia*, *coma-vigil*, and muttering delirium. In favorable cases improvement begins on the third or fourth day, from which time the symptoms gradually subside. Convalescence may be said to be established when restful sleep can be obtained; this is followed by a desire for food. In unfavorable cases the patient may pass from a typhoid state into exhaustion and death, or may die suddenly either during a paroxysm of cardiac failure or from some complication, as cerebral hemorrhage or pneumonia.

**Diagnosis.**—The condition of persons found dead-drunk is seldom mistaken for any other. The reverse more often happens, and in this way *apoplectic* and *uremic comas* may be diagnosed as alcoholic coma. Cases picked up in the street in a state of apparent unconsciousness

should be carefully tested in this regard. Instances in which, as the *postmortem* examination subsequently has shown, cerebral hemorrhage has followed a drinking-bout, render the diagnosis more difficult; in such the patient should be given the benefit of the doubt and handled as though the case were one of apoplexy. An important early step is to ascertain whether the coma is complete, or whether the patient can be roused by shouting in the ear, by applying ammonia to the nostrils, or, better still, by pressing, with gradually increasing firmness, over a sensitive spot, as the supraorbital notch; if the unconsciousness is alcoholic, he will come to his senses, if only for a moment. Abstemious apoplectics have been known to stagger and talk thickly, like drunken men (Kerr), and have been arrested and taken to a police-station instead of to a hospital. *Congestion* and *lobar pneumonia* affecting the bases of the lungs should be looked for, as they are common causes of death in drunkards. A table giving the principal points in the differential diagnosis will be found under *Uremia* (*vide* p. 965).

The diagnosis of chronic alcoholism is made from the history, and from the muscular tremors (worse in the morning), vomiting, mental restlessness, "mendacity," and involuntary "lying" (Kerr). The condition may resemble general paralysis, and if the habits of the patient are kept secret it may be very difficult to differentiate these affections. A prominence of disorder of the digestive tract usually points to alcoholism. Nervous excitement, tremors, fear, wakefulness, and the distinctive physiognomy are more evident in chronic alcoholism, even when general paralysis has been caused by alcohol, which is apparently the case. *Paralysis agitans*, *locomotor ataxia*, *epilepsy*, and *nervous dyspepsia* may also be mistaken for chronic alcoholism by the unwary.

*Delirium tremens* is distinguished by the history, by the restlessness, delirium, hallucinations, tremors, and terrors. *Mania-a-potu* differs from the preceding mainly in its usual association with acute alcoholism in neurotics, in the muscular contractions, the furious mania, and convulsive movements. The delirium of *apical pneumonia* that obtains in some cases (as well as in meningitis) must be thought of in the diagnosis of delirium tremens. The diagnosis of *alcoholic neuritis* from other conditions simulating it will be found elsewhere (*vide* p. 1029).

**Prognosis.**—In acute alcoholism the prognosis is favorable in private, manageable cases. Many of the cases brought into hospitals are affected also with pneumonia, and usually die. The tissue-changes in chronic alcoholism are so profound, and they affect such delicate and vital tissues, that when the alcohol-habit thus becomes fixed permanent recovery never takes place. The treatment appropriate for the inebriate and forced abstinence from alcohol relieve many of the symptoms and some of the debility, but relapses are all too common and are almost certain to occur. Insanity and paresis are not infrequent terminations of chronic alcoholism. Many complications are apt to supervene, as Bright's disease, epilepsy, melancholia, fatty heart, pneumonia, and thrombosis. Alcoholic neuritis often clears up upon withholding alcohol and stimulating the peripheral nerves both by appropriate drugs and external remedial measures. Recovery from delirium tremens is dubious in cases of severe injury, inflammatory troubles, or infections.

**Treatment.**—In cases of acute drunkenness, which are only too



commonly met with, nothing special is required except to prevent the ingestion of any more alcohol and to allow the patient to sleep until the elimination of the poison is more or less complete. The effects of the intoxication, in the general depression, headache, anxious and irritable stomach, and various functional visceral and nervous disorders, may need careful corrective and sustaining treatment for a week or more. The diet should be light and nutritious. Aperient waters, hot baths, with liquor ammonii acetatis frequently repeated, and a combination of dilute mineral acid and bitter tonics (nux vomica, gentian), are also indicated.

In profound cases of alcoholic coma, convulsions, or mania-a-potu no alcohol should be given. Trite though this injunction may seem, it is important to emphasize this statement, so that the physician may be sure to counteract a popular impression that the giving of more alcohol will cause a mania to subside *permanently*, and to guard against the smuggling of liquor to the patient by his misguided friends. It is often necessary to empty the stomach at once when collapse is imminent by the use of the stomach-tube or -pump, washing out the organ with hot water, to which ginger or cinnamon has been added. To this end emetics may be used—viz. ipecac or apomorphin, hypodermically (gr.  $\frac{1}{8}$  to  $\frac{1}{6}$ —0.008–0.0108). The external application of warmth, friction, artificial respiration, faradism to the phrenic nerve, ammonia- or amyl-nitrite-inhalations, and hypodermics of atropin, strychnin, and digitalis, may all be tried. Hot rectal enemata or a calomel purge if the stomach will tolerate the drug should be used early. The maniacal attacks may be treated by hypodermics of morphin and hyoscin, and by such sedatives as chloral, bromids in large (3j—4.0) doses, and rarely such hypnotics as paraldehyde, trional, chloralamid, and the like. Indeed, it is very important to secure sleep as soon as possible. An excellent formula in cases of medium severity is:

R $\bar{y}$ . Sodii bromid.,	3j (32.0);
Tr. capsici,	3j (4.0);
Tr. digitalis,	3ss (2.0);
Elix. simplicis,	q. s. ad 3ij (64.0).—M.

Sig. 3j (4.0) every two or three hours, in water.

As soon as some quietude and sleep have been obtained, it is in order to administer concentrated food in an easily assimilable form.

The treatment of *chronic alcoholism* is more often best conducted in “homes” for inebriates, in hospitals, and similar institutions. At the outset there must be an “unconditional surrender” in the use of alcohol. Its withdrawal should be enforced at once in many cases, and very rapidly in all others, according to the judgment of the physician as to the psychic and physical condition of the patient. Substitutes for alcohol are the strong fruit-juices, as hot lemonade or hot ginger, and cardamom tea often is useful. Coffee, milk, cocoa, and hot broths are also to be recommended. The diet should be carefully increased in nutritive strength as the gastric irritability diminishes. Sometimes such sedatives to the stomach as the bismuth-preparations, effervescent alkaline drinks, and lime-water may be indicated. Peptonized food is often well borne at first in cases in which gastric distress is marked. Nutrient enemata are seldom required, but should be resorted

to in the gravest cases, particularly during the states of alcoholic dementia. The general health must be looked after by placing the patient in the best of fresh air, exercise, cold and warm bathing, by mental and social occupation, and by diversion. When the craving for alcohol is hereditary and intense, seclusion in an inebriate-house or some similar institution is often necessary for a long time, inasmuch as the danger of lapsing into the former drink-habit is so common in these cases.

The insomnia of chronic alcoholism may be met temporarily by the use of large doses of bromids, chloral, hyoscin, or sulfonal. Morphin may be indicated at times, but should be used with great caution in order to avoid adding the morphin-habit to that of alcohol. Perhaps the best single agent to use in counteracting the symptoms of chronic alcoholism is strychnin, either as the nitrate or sulphate, hypodermically and by the mouth; iron, arsenic, the hypophosphites, dilute phosphoric acid, quinin, *avena sativa*, and the like are often useful adjuvants in the tonic treatment. Atropin, hypodermically, may also be recommended when vascular dilatation and weakness are prominent. A "substitute" for alcohol, both for its local and mental effects may be prescribed to meet the occasional cravings. Tart fruits (as oranges and lemons), coffee, hot malted milk, ginger, gentian, and capsicum infusions may be tried. Tinctures should not be given in this form for obvious reasons. Sweating and purging the patient, and the administration of bromids, chloral, and gelsemium for a day or two in advance, may avert a "drink-storm" or the periodic cravings for alcohol that may be expected by prodromal manifestations. Sometimes, however, as in the sudden outbursts of dipsomaniacs, there is no time to institute their treatment. It is claimed that hypnotic suggestion will abolish effectually the ardent desire for alcohol in a certain number of neurotic cases of alcoholic inebriety. Temperance revivals may be said to do permanent good only in those similar neurotic cases that are fortunately impressionable with appeals by total-abstinence orators, but, in order to maintain the reformed drunkard's pledge it is often necessary that interested persons continue to watch, guide, and inspire him, in order that a weakened will may not precipitate a cyclic lapse into his old habits.

In all cases the treatment will be incomplete unless the highest part of the patient's nature receives due attention throughout. The reason must be enlisted in the treatment, and this is best attempted by sound teaching concerning the fallacy of the prevalent belief in the virtues of alcohol as a beverage.

No pains should be spared to impress upon the patient the need of a persistent abstinence from all intoxicants as long as he lives. All the influence of culture, music, and the fine arts, of high-toned morality and pure, undefiled religion, should be enlisted to strengthen self-respect and to fortify volition and inhibition. Moral regeneration may thus in certain cases check indirectly the physical and mental degeneration, but it cannot efface the consequences of the alcoholic poisoning which it represents.

**Delirium tremens** requires firm but tactful isolation and vigilant nursing. All alcohol should be withheld. If stimulation is needed, aromatic spirits of ammonia, strychnin, and atropin, with bland hot

drinks and broths, may be administered. Easily digested and nutritious food should be given to support the strength. Sleep must be procured by such means as are mentioned above in the treatment for *mania-a-potu*. The dosage required, however, is usually not as great, but must be kept up longer than in the maniacal condition. Cardiac weakness may need such stimulants as digitalis, strophanthus, and the ammonium salts. After the attack subsides, tonic doses of strychnin, chirata, gentian, asafetida, calumbo, and iron, together with graduated exercise out of doors, are to be employed. Turkish baths, industrial occupations, and the like are indicated to conserve the patient's strength and thus fortify him against yielding to temptation and a morbid appetite.

#### GINGER AND COLOGNE-WATER INEBRIETY.

Habitual drinkers of alcoholic ginger, capsicum, and lavender preparations, and eau-de-Cologne are practically alcohol-habitués or inebriates. They drink these liquids for the alcohol that is in them. The so-called essence of ginger (Jamaica ginger), which contains considerable alcohol in some of its preparations, is often used primarily for relieving an attack of "cramps" or "colic," and if frequently repeated, can readily induce a morbid habit of "ginger-drinking." In other cases the craving for alcoholic indulgence (often hereditary), may have been aroused by a social glass of wine, but, from a sense of shame the desire has been kept secret, and gratified by drinking eau-de-Cologne, lavender essence, or even tincture of capsicum. Perhaps many more such cases exist, and especially among neurotic women in good circumstances, than are usually recognized.

#### MORPHINISM.

(*Opium-inebriety*.)

**Definition.**—A chronic intoxication, due to the habitual use of morphin or of opium in some other form (*opiumism*).

**Pathology.**—In cases of death from acute or chronic opium- or morphin-poisoning there is nothing distinctive in the pathologic appearances. In acute cases vascular congestion of the brain and membranes has been noted; but even in chronic cases the tissue-degeneration and fatty and connective-tissue proliferations that are characteristic of alcoholism, are practically absent. Decided lesions are usually traceable to associated affections. The principal anatomic changes are those due simply to malnutrition. Thus, we have the emaciation and the shrunken appearance of cerebral anemia, and pallor and atrophy of the cardiac muscle and of the vascular walls. The dried and wasted structures, due to tissue-starvation, are quite a contrast to the fat-infiltrated or degenerated, cirrhotic, and inflamed tissue of alcoholic inebriety. Direct destruction of parenchymatous cells is more evident in the later.

**Etiology.**—The climate, country, and nationality have a certain disposing influence in the development of opiumism and morphinism. In the opium-growing parts of Asia, as in China, India, and Persia, where the climate is warm, enervating, and conducive to physical and



moral abandonment during the greater part of the year, and in Turkey also, opium-eating-and-smoking habitués are as numerous as alcohol-habitués are in Europe and America among the Caucasians. Morphine is more common here than is opiumism, except among the poverty-stricken.

Women are more commonly the victims of morphinism than men, except physicians and druggists as a class. Mattison has found 70 per cent. of his opiate patients to be medical practitioners. Many contracted the habit by using morphin for severe chronic neuralgia, insomnia, and the like. Indeed, pain and sleeplessness have been the principal source of this drug-habit.

*Ennui* and an idle spirit of irritation and adventure among the sensation-loving and luxurious sometimes sow the seeds of an indulgence in narcotics that bring forth fruitage in the form of a fixed, morbid, and uncontrollable craving for constant satisfaction, and a consequent physical, mental, and moral decline.

The incautious prescribing of morphin and the too ready hypodermic use of the alkaloid by physicians in treating various cases of pain are not infrequently the cause of morphinism. Overwork of the brain, great business or social strains, prolonged worry and anxiety either with or without work, insomnia, remorse, idleness, and secret vices, are the most common predisposing agents of the morphin-habit.

Paregoric, laudanum, chlorodyne, and "soothing-syrup" are drunk to a frightful extent in large cities among the poor and miserable, and cause great disturbance of the health of the habitués.

**Symptoms.**—These may be in abeyance for some time, while the habit is forming and the doses are still slight. As the craving increases, the dose and its frequency increase to keep pace with the desire. Anemia gradually develops, with sallowness of the skin, wasting of the features and body, languor, weakness, functional deterioration, mental depression, anorexia, restlessness, insomnia, tremors, irritability, shyness, dilatation of the pupils (except when under the influence of the drug), and a characteristic propensity to lying. Cardialgia is often complained of by those who use opium pretty constantly. The associated vices of opiumism are less violent and inflammatory than those of alcoholism, and more secretive and speculative, such as gambling and sexual perversions. Itching is frequent, and especially after taking the opium or morphin. Attacks of chills, followed by pyrexia, with delirium and transient albuminuria (renal congestion) occur in some cases. Diarrhea and dysentery have been observed in some instances. There may be also disturbances of the visual muscular apparatus. Sufferers from painful carcinoma in whom opium or morphin is required for steady use do not become, except in rare cases, true morphinomaniacs.

The *course* of morphinism is that of a progressive asthenia, in which cardiac palpitation, dyspnea, abdominal and muscular cramps, trembling, fear, sleeplessness, mental confusion, melancholy, slovenliness, and moral obtuseness come on. Some women, known to be kleptomaniacs, have been found to be secret opiumists. Sexual impotence in the male, and amenorrhea and abortion in the female, are common results. The skin is wrinkled, dry, and harsh, and may show numerous needle-scars and abscesses in those addicted to the hypodermic use of the drug. The

termination is the direct result of the extreme debility or marasmus or of some intercurrent affection.

The **diagnosis** must be made from the history. When the latter is wanting because of a lack of veracity or deception, *chronic alcoholism* may have to be differentiated from opiumism. The more open and often periodic habits of the alcoholic habitué, and the general aspect of the physical and mental and complicating conditions, usually show marked differences between the two drug-intoxications.

**Prognosis.**—The likelihood of a cure is exceedingly remote. On the other hand, under proper conditions much relief may be given the morphinomaniac, and although the habit may be suspended only for a time, life may thus be prolonged for years.

The **treatment** is manifestly difficult and unpromising. Institutional isolation, rest, diversion, watchful care, regular and studied feeding, baths, and graduated exercise in the open air as far as possible, but under surveillance in order to prevent the smuggling of opium, morphin, or compound preparations containing either, are the most efficient measures. As to the manner of withdrawing the narcotic, much care, judgment, and tact form a *sine quâ non* in the treatment. A sudden and absolute stoppage of the use of the drug sometimes leads to great distress, and even to collapse ("abstinence phenomena"); it is, therefore, not to be recommended, as in chronic alcoholism. On the other hand, the too gradual withdrawal is torturing. A middle course, the "rapid-gradual method" of Erlenmeyer, is usually resorted to, in which the reduction of the quantity of morphin or opium to nothing occupies but a week or ten days. Various substitutes have been recommended that generally prove not to be substitutes at all, but simply act in a symptomatic way, and may lead to another habit as bad if not worse. Such drugs as cocaine, hyoscyamus, belladonna, bromids, and chloral have thus been used.

In the symptomatic treatment of the morphin-habit moderate doses of bromids, with cannabis indica and some such vegetable bitter as gentian, may prove useful in allaying the nervous irritability and restlessness at night. Sulfonal is a good hypnotic in these cases. Cathartics, stomach sedatives alternating with tonics, concentrated foods, massage, hot and cold bathing, electricity (general galvanization), and "complete control over the patient" are usually indispensable adjuncts in the treatment after the withdrawal of the opium or morphin. Cardiac stimulants, strychnin and physostigmin salicylate (gr.  $\frac{1}{100}$ —0.0006) hypodermically, have been recommended recently as important in counteracting the functional depression of these habitués. Industrial activity, and mental and social diversion, aid in maintaining any improvement made and in rendering the patient less liable to a relapse.

#### PLUMBISM.

(*Chronic Lead-poisoning; Saturnism.*)

**Definition.**—A chronic intoxication due to the slow absorption of lead, either industrially or accidentally.

**Pathology.**—The principal lesions are found in the muscles, peripheral nerves, liver, kidneys, and mucous membranes. The affected mus-

cles are wasted, pale-yellow in color, and, in advanced cases, show a marked fibroid growth. The vessels in the muscles also reveal arteriosclerosis. The peripheral nerves are affected with a parenchymatous neuritis, and are especially involved, with degenerative changes in the nerve-endings in the muscles. The nearer we approach the spinal cord along the course of an affected motor nerve, the less marked are the changes, although in some cases a very slight involvement of the anterior nerve-roots has been noted. The cord is usually normal.

In the brain, the pathologic changes scarcely warrant us in attributing lead encephalopathy to them. Aside from a slight meningitis and arteriosclerosis of the cerebral blood-vessels here and there, with a corresponding connective-tissue growth and capillary hemorrhages, the evidences of lead-poisoning are practically *nil*. Cerebral symptoms are most probably the outcome of functional disturbances. The liver and kidney show parenchymatous atrophy and cirrhosis.

**Etiology.**—(a) *Personal susceptibility* to lead-poisoning is greater in some people than in others, all other things being equal. (b) Plumbism is more common in *adults* than in *children*, because of greater exposure. (c) *Sex*.—Women are more susceptible than men. (d) *Occupation* is the most frequent cause of lead-intoxication. Workers in white lead (plumbic carbonate), red lead, and litharge, all of which substances are used as paints, are especially to be mentioned as liable to saturnism. Among the most common industrial causes are the following: painting, plumbing, lead-mining, rolling sheet-lead, pottery-glazing, type-founding and setting, shot-making, dress-making (in which lead-dyed silk thread is used and the ends bitten off), lace-making, glass-grinding, and calico-printing. (e) An accidental source of lead-poisoning is found in the contamination of food and drink. Men employed in the manufacture of white lead and eating lunches in dusty work-rooms may also suffer from plumbism in this way. Drinking-water stored in lead-lined cisterns and passed through lead pipes is frequently contaminated, especially if the water contains a slight amount of acid. Flour, bread, biscuit, candy, butter, and milk may cause poisoning by adulteration with lead chromate, used to give a rich, yellow tint to these articles; and tobacco wrapped in lead-foil has, less commonly, resulted in symptoms of saturnism.

The *absorption* of the lead takes place mainly through the gastrointestinal tract, especially through the lungs, and much less through the skin. It may be deposited in most of the soft tissues and viscera, but especially in the nerves, muscles, and liver. *Elimination* takes place through the kidneys, and probably, though in very slight quantities, with the bile and saliva, and through the skin.

**Symptoms.**—Depending upon individual susceptibility, it may be months or years before the first manifestations appear. *Anemia* is an early and marked symptom (*saturnine cachexia*). There is a moderate reduction of the corpuscles and of hemoglobin. The general nutrition is poor.

The characteristic *blue line* at the borders of the gums is rarely absent, especially in those who are not scrupulous in their attention to the teeth. It is, as a rule, most distinct at the roots of the lower ca-



nines and incisors, and is formed by a deposition of lead sulphid. Bluish patches may also be met with.

*Colic* is very common and is also characteristic. The pains center around the navel, and are quite severe and griping. They are associated with retraction and rigidity of the abdominal walls, and with obstinate constipation. The pains are paroxysmal, may be referred at times to the epigastrium, and may be accompanied by vomiting. Between the paroxysms a dull pain usually exists over the whole abdomen. During the attacks of colic the pulse-tension is increased and cardiac action lessened.

*Paralyses* are common symptoms, and may either be acute, subacute, or chronic in nature. Although usually localized palsies, they are sometimes generalized. The most characteristic lead-palsy is that known as *wrist-drop* (see also Multiple Neuritis, p. 1029).

Both fine and coarse *tremors* occur, though not so commonly as in chronic mercurial poisoning. They usually begin in the hands and arms, are rather constant, and are aggravated by *voluntary effort* and emotional excitement.

*Cramps* in the affected muscles and about the joints (*lead-arthralgia*) are occasionally noted. Slight anesthesia, especially in cases of wrist-drop, is sometimes detected here and there, but may in certain instances be due to saturnine hysteria.

The *cerebral symptoms* are important. The phrase "lead encephalopathy" includes such manifestation as delirium and coma, neuro-retinitis, aphasia, convulsions, hemiplegia, amaurosis, hysteria, and insanity. The delirium and coma are the commonest brain-symptoms, and may come on suddenly with tremors and hallucination. Epileptic convulsions are often severe. Hemianopsia has been observed. Mania and melancholia occur in cases of mental unbalancing, and hysteric outbreaks are seen in girls. Intense headache is not uncommon. "Saturnine gout," so called, is described as a result of chronic plumbism. The kidneys are contracted, the heart is hypertrophied, and arterio-sclerosis is marked, with a diminution in the excretion of urea and uric acid. The pulse-tension is increased. These evidences show a similarity to gout, and favor the development of uratic deposits in the joints, but they are the effects of "mineral," and not of essential or true gout. Lead may be discovered in the urine by laying a strip of magnesium in it and noting the deposit of metallic lead if present (Von Jaksch). Abram asserts that the addition of a solution of ammonium oxalate (1 gm. to 150 c.c. of water) facilitates the test.

**Diagnosis.**—The history of exposure to lead-poisoning is usually clear in those working the metal in its various forms. *Accidental origins* of saturnism are often obscure and very difficult to trace, although if the characteristic wrist-drop, the blue gingival line, colic, and cachexia be present, the diagnosis is readily made.

*Alcoholic paralysis* of the lower extremities may be differentiated by the history, the greater prominence of sensory symptoms, and by the absence of the blue line on the gums.

**Prognosis.**—In the absence of the graver nervous, arterial, and renal symptoms, the prognosis is good. When there is profound paralysis, with reactions of degeneration, and especially in primary

atrophy of the muscles, the prognosis is generally bad. In the severe encephalopathic forms, and in cases in which marked arteriosclerosis and renal cirrhosis are manifested, the prognosis is unfavorable, but depends upon the extent of damage done.

**Treatment.**—The prevention of plumbism is difficult in lead-working establishments, owing to the carelessness and indifference of both employers and employees, and to the lack of any adequate antidote during exposure. Rigid cleanliness is absolutely necessary, especially of the hands and nails and before eating. Means to allay dust should be regularly and constantly employed. Milk and sulphuric-acid lemonade have been recommended for use by workers in lead, for their supposed antidotal effects. As perfect ventilation as possible should be secured, and respirators are in use in some lead-works, being worn as “snouts.” Potassium iodid should be given in chronic plumbism, beginning with small doses (gr. iii-v—0.1944–0.324), given preferably in milk, after meals.

In *lead colic* hot applications to the abdomen and hypodermic injections of morphin and atropin are often indicated. Efficient doses of Epsom or Glauber's salts are used to combat the constipation. Given in combination with dilute sulphuric acid (in order to form an insoluble lead sulphate) and with belladonna, the best and speediest benefits may be obtained thereby.

Iron for the anemia, strychnin and galvanism for the paralysis, lithia-water for the renal deterioration, and nitroglycerin or sodium nitrite for the arteriosclerosis (enough to relieve increasing tension) are the symptomatic items of treatment that are usually indicated. Rarely, hopeless cases of saturnine encephalopathy need to be sent to asylums for the insane.

#### ARSENICISM.

(*Chronic Arsenic-poisoning.*)

**Definition.**—A chronic intoxication resulting from the gradual absorption of arsenic.

**Pathology.**—The peripheral nerves show a degenerative neuritis, and the anterior horns of the spinal cord may be similarly affected.

**Etiology.**—The causes of arsenicism may be habitual, industrial, medicinal, or accidental. The individual predisposition to arsenic-intoxication varies in different persons. A neurotic diathesis usually underlies the habit of “arsenic-eating” in those who crave the drug for its alleged exhilarant or narcotic effects (*arsenic inebriety*). Not a few women suffer from chronic arsenicism as the result of the ingestion of arsenic “to improve the complexion and brilliancy of the eye.” Men employed in arsenic-works of various kinds often suffer from the chronic poisoning. For example, miners and smelters of arsenic pyrites, dyers and wall-paper workers using Scheele's or Schweinfurth's green, artificial-flower makers, shot-makers, glass-workers, and taxidermists, are all liable on account of their occupations. Sometimes the medicinal use of moderate doses of arsenic, as in Fowler's solution, even for a short time, may in very susceptible persons induce arsenical paralysis (Putnam; Osler). Accidental arsenicism may come from living in rooms where wall-paper, carpets, colored paper ornaments, toys, or

curtains are contaminated with arsenic anilin dyes; this does not occur so frequently as years ago.

**Symptoms.**—There are anemia, loss of flesh and strength, dryness and irritation of the mucosæ, of the eyes, nose, throat, and upper respiratory tract. Anorexia, nausea, and diarrhea indicate the presence of a gastro-intestinal catarrh. In some cases, milder than others, the fat is well preserved. Slight puffiness of the eyelids or eyebrows may occur, and some epigastric distress may be complained of. Marked conjunctivitis, occasional dysenteric attacks, loss of the hair, and numbness and tingling in the extremities form a commonly observed symptom-group. Cutaneous symptoms may appear, as pigmentation ("arsenic-bronzing"), and eczematous, herpetic, urticarial, and pemphigoid manifestations. Albuminuria with casts and blood mark the renal irritation that sometimes occurs.

The most characteristic evidence of chronic arsenic-poisoning is seen in the gradual increasing diffuse or multiple neuritis. Differing from lead-palsy, the leg-extensors and the peroneal group of muscles are involved first, although the arms may also become affected later (*vide* Multiple Neuritis, p. 1030). Contractions in the lower and a fine tremor of the upper extremities are apt to occur. Arsenic-poisoning may also cause headache, vertigo, melancholia, and hysteria. The drug is eliminated by the kidneys and may be found in the urine. Sometimes a great toleration of arsenic is observed in workmen and habitués, the only evidences being a clear, sallow, waxy complexion, a gloomy expression, and some dyspepsia, perhaps, as in the well-known Styrians.

**Diagnosis.**—This is not difficult, when once the source of the poisoning is determined. The clinical appearances are distinct from *lead-intoxication*, especially in the mode of progress of the paralysis, and in the more marked sensory symptoms combined with the motor-disturbances of arsenicism.

The **prognosis** is favorable in most cases in which removal from the exposure to the influence of arsenic is possible. A few cases die from the great general debility.

**Treatment.**—Abstention from the use of arsenic for cosmetic purposes, avoidance of its influence in the arts, care in its medicinal administration, and prophylaxis as regards the possible or discovered sources of contamination, form the first considerations in the treatment. Elimination of the arsenic may be promoted by the use of potassium iodid and purgatives. Gastro-intestinal and other irritations must be met by appropriate sedative remedies. The neuritis and palsies require—as soon as the tenderness and pain subside—massage and electricity. Judicious and wholesome alimentation and tonics are indicated.

#### MERCURIALISM.

(*Chronic Mercurial Poisoning.*)

**Definition.**—A chronic intoxication caused by the habitual ingestion, or combined industrial absorption of mercury, in susceptible individuals.

**Pathology.**—No marked pathologic changes have been noted in human beings, aside from the evidences of oral, gastro-intestinal, and



renal irritation and inflammation. It is not improbable that the cerebral cortical areas suffer more from metallic irritation than do the spinal or peripheral nerve-tissues.

**Etiology.**—Some persons are much more easily mercurialized than others. (a) *Salivation* and *stomatitis* from the therapeutic use of mercury form a variety that is not infrequent in these days. (b) *Industrial origin.* The chief cause of chronic mercurialism is the inhalation of the vapor of the metal by artisans in the industries in which it is used. Thus miners and smelters and those engaged in making mirrors, barometers, thermometers, amalgams, felt hats, vermilion-pigment, and artificial teeth sometimes suffer from chronic mercurial poisoning. It should be pointed out here that mercury is volatile at ordinary temperatures, and is absorbed into the blood through the lungs, digestive tract, and skin. Calomel vapor-baths have caused poisoning in a few cases. (c) Purely *accidental* mercurialization also occurs. (d) Women and children are more susceptible to the action of mercury than men. In all cases the mercury exists in the tissues as an albuminate.

**Symptoms.**—There are anemia, emaciation, gastro-intestinal disorders, stomatitis, salivation, maxillary necrosis, ulceration of the gums, loosening of the teeth, fetor of the breath, marked tremors, and paralysis. The oral symptoms are not as prominent, however, as in acute mercurial poisoning. The hair falls out, the nails become brittle, and pigmentation of the skin is seen.

The *tremor* is characteristic. It is first felt or noticed in the tongue and lips, is usually fine, later coarse and choreiform, and spreads gradually throughout the muscular system. It is aggravated by voluntary effort, and may cease during sleep in mild cases. Speech is altered. Hysteric tremors may also exist. Great irritability and restlessness are common. Aphasia, hemiplegia, hemianesthesia, and peripheral neuritis with palsies, occur. There is no atrophy, nor are the reactions of degeneration present in the paralyzed muscles. Severe pains may be present in the extremities, including the joints, and grave cerebral symptoms occasionally develop (stupidity, headache, loss of memory, insomnia, hallucinations, delirium, coma, convulsions, and confusional insanity). Albuminuria with anasarca may occur. The effects of chronic hydrargyris in women upon their offspring are also important, the children being rachitic, weak, sickly, and prone to tuberculosis.

**Diagnosis.**—The history, the characteristic tremors, paresis, and mental irritability are significant. In the absence of a history of exposure to mercury, the differentiation from *progressive general paresis*, *disseminated sclerosis*, or *paralysis agitans* may be more or less difficult.

**Prognosis.**—Recovery is common upon the removal of the source or on removing the patient from the source of the poisoning. Fatal terminations rarely ensue, and then in cases of mercurial encephalopathy of a grave type and with a tendency to idiocy.

**Treatment.**—Prevention of further poisoning is imperative, elimination is to be promoted, and the symptoms are to be met as they arise. Potassium chlorate, with the tincture of myrrh, and astringents are useful for the occasional stomatitis and salivation. Potassium iodid, and also sulphur baths, may be used to aid in the elimination of the mercury. Iron, cod-liver oil, good food and fresh air, and a free activity

of the emunctories are of positive value. Electricity may be resorted to for the paresis.

#### FOOD-INFECTION AND PTOMAIN-POISONING.

In recent years there have been reported an increasing number of cases of serious illness that have been traced to infected and contaminated food. Undoubtedly many such instances are now brought to notice that in former times were attributed to other causes, or that were not diagnosticated because of a lack of knowledge. On the other hand, the increased consumption of canned and preserved meats has certainly augmented the liability to poisoning from these products, as the reports of cases show. Lack of care in the inspection and selection of the meats, uncleanness, and sometimes unscrupulousness, in their handling and preparation, must result in infection, putrefaction, and toxicity. The infection of the food may be due to (1) disease of the animal or plant from which the food is derived; (2) microbic inoculation of the food after derivation and before ingestion by human beings; (3) infection by toxicogenic bacteria, and the presence of ptomains or toxalbumoses. The transmission to man of such affections in animals as tuberculosis, anthrax, glanders, and pleuro-pneumonia, by eating the infected meat, has been sufficiently proved. Again, meat and milk may become infected, before being ingested by the patient, by pathogenic microorganisms, as of typhoid fever and diphtheria, or from the production of toxins owing to the action of non-pathogenic putrefactive microorganisms. A great many instances of food-infection, particularly of meat and milk, have been shown to be due to the presence of saprophytic germs, this happening even when the articles of food have been obtained from healthy stock and have been kept free from specific pathogenic bacteria. It is not, however, the saprophytes themselves in all cases, but the poison developed in the food before it is eaten or formed in the body afterward, that produce the symptoms and sometimes death. According to Novy, some of the saprophytic bacteria with which food is infected outside of the body, under certain conditions, are capable of living in the body as parasites, especially on dead matter, and there become toxicogenic.

The chronic poisons or ptomains resulting from the action of the saprophytes in foods are called "putrefactive alkaloids;" those bacterial products of a proteid nature are called "toxalbumins" or "toxalbumoses." The latter, according to Vaughan, are more frequently present in infected foods. They are all absorbed from the digestive canal.

**Poisoning by Infected Milk and Milk-products.**—It is now well known that the cause of the high mortality-rate among infants in hot weather is traceable directly or indirectly to the "summer diarrheas" in children fed artificially, wholly or partially, with milk infected by numerous varieties of saprophytic germs and thus poisoned by ptomains, such as tyrotoxin. This special chemical poison has been isolated by Vaughan, and discovered by him in cheese. It has also been found in ice-cream, frozen custards, and cream-puffs, and has caused poisoning-symptoms mainly of acute gastro-intestinal inflammation, "constriction of the fauces," nausea and vomiting, sharp, griping intestinal

pains, headache, thoracic oppression, chilliness, dizziness, and sometimes purging, followed by relief in mild cases. In the severe and long-continued forms, however, exhaustion may supervene, with subnormal temperature, coma, collapse, and death in the graver cases. No chemical or physiologic antidote is known. Elimination may be assisted, and stimulation is needed. Irrigation may be employed for the former in both stomach and bowels. Strychnin, nitroglycerin, atropin, and the aromatic spirits of ammonia are most effective as stimulants.

**Meat-poisoning.**—Various tainted meats, as mince-meat “warmed over,” veal pie, carelessly-kept chicken salad, badly-preserved and canned meats, partially-decayed sausages (*botulismus*) have caused violent symptoms of poisoning. Diseased raw and partially-cooked meat has also been eaten with disastrous results. It should be borne in mind that even prolonged cooking fails to destroy the toxic action of certain ptomains in infected meats; also, that meat that has been cooked and kept under certain conditions may become infected with bacteria as well as when it is raw. On the other hand, bad, putrid meat has been known not to cause toxic symptoms.

The *symptoms* caused by the poisoning are—“(1) those due to a true infection; (2) those due to simple poisoning” (Mann). Cases of the former group run the usual course of an infectious disease, often simulating typhoid fever. Those under the second division manifest the symptoms of a violent gastro-enteritis, with vomiting, intense colicky pains, purging, fever, accelerated pulse, nervous prostration, great muscular weakness, and cramps in the calves of the legs. Often a subsequent subnormal temperature, extreme depression, convulsive movement, vertigo, dimness of vision, dyspnea, somnolence, great soreness of the mouth, collapse, and sometimes death supervene. The mortality-rate varies from 15 to 55 per cent. of all the cases.

The *treatment* is largely eliminative, symptomatic, and supportive. The prophylactic measures, private and public, are generally obvious.

**Poisoning by Fish (*Ichthyismus*) and Shell-fish.**—Many instances of this serious form of intoxication have been produced. The fish may contain certain poison-glands, ovaries, etc. Especially is this true of certain species known in Japan, one of which is believed to cause the disease called “Kakke,” which prevails during the summer months in Tokio. A certain species of fish (*Clupea venenosa*) inhabiting the West Indian waters is supposed to be always poisonous, although the source or true character of the poison is doubtful. In Russia, many cases of ichthyismus have resulted from eating both the fresh and preserved sturgeon and salmon meat that are affected with an infectious disease peculiar to the fish. In Germany and other parts of middle Europe a severe form of gastritis called “Barbencholera” follows the eating of sick barbels.

The use of tainted preserved and canned fish, eels, oysters, mussels, crabs, lobsters, and the like, is more frequently the cause of symptoms of poisoning, however. Brieger's *mytilotoxin*, the active poison formed in some mussels, and the eating of which at Wilhelmshaven caused several epidemics, is probably developed only under certain favorable conditions of saprophytic infection. Devilled crabs, lobsters, and salad have also caused severe gastro-enteritis because of contamination with germs pro-



ducing ptomains. Oysters have been accused of conveying typhoid infection (*vide* p. 26). The *symptoms* of fish- and shellfish-poisoning are variable. Sometimes marked cerebro-spinal manifestations predominate, with convulsions and paralysis. Dryness and constriction of the throat, dizziness, labored respiration, disturbed vision, jerky speech or aphonia, perhaps rapid pulse, loss of coördination, numbness, coldness of the extremities, dilated pupils, paresis, collapse, and death within a few hours, may ensue.

Other cases have a pronounced gastro-intestinal or choleraic group of symptoms, with nausea and vomiting, pain, tenesmus, and mucous and bloody stools. In some of them marked cutaneous irritation is shown by erythema, great heat and itching, urticaria, and swelling. Dyspnea, lividity, and sometimes delirium, have also been noted. The *prognosis* is grave in many instances. The *treatment* is similar to the above—namely, emetics, purgatives, enemata, and lavage. The indications are to be provided for as they arise.

#### GRAIN- AND VEGETABLE-POISONING.

**Ergotismus.**—Epidemics of ergotism have resulted from the continued use of meal made from contaminated grains grown on virgin soil. The parasite (*claviceps purpurea*) is a fungus that infests rye and other grains; it does not, however, grow readily where the soil is well cultivated, and epidemics of ergot-poisoning are much less frequent than formerly, if we except certain places in Spain and Russia. According to Kobert, three poisonous substances are found in the ergot: ergotinic acid, sphacelinic acid, and cornutin. The first of these is not poisonous when taken into the stomach; the second is supposed to cause gangrene; and the last produces grave effects on the nervous system, and is found only in fresh ergot, hence the greater prevalence of nervous manifestations in sickness that breaks out soon after harvest.

The nervous symptoms are remarkable for their convulsive characteristics (*ergotismus convulsivus*). Prodromes of weakness, tingling in the extremities, and headache may exist for several weeks before the spasms come on. The formication increases, and cramps and contractures, with flexed wrists and extended feet and toes, seize the patient. In severe cases epileptoid convulsions occur and may prove fatal. Delirium and, in very chronic cases, dementia may supervene. Recovery is slow, and the contractures may persist for some time, with muscular atrophy and anesthesia. In some interesting instances there may appear nervous symptoms resembling locomotor ataxia ("ergot tabes"), owing to posterior spinal sclerosis. Abortion results in pregnant women.

Gangrenous ergotism (*ergotismus gangranosus*) is characterized by dry gangrene of the hands and feet, usually of the fingers and toes. Before the gradual blackening appears, there may be formication, pain, spasm, numbness, and coldness. As mortification and the line of demarcation progress, the parts drop off bit by bit, and fever may attend the sphacelation. Pneumonia (septic) may sometimes complicate this malady. The fatality has been considerable in some epidemics. The *treatment* of ergotism is entirely symptomatic.

**Maidismus or Pellagra.**—This is a chronic nutritional disturbance

due to poisoning from eating contaminated corn-meal bread. The disease prevails extensively among the poorer classes in Lombardy, Spain, and southern France. The origin of the infection of the maize is said to be bacillary, the latter causing putrefactive or fermentative changes in the fresh, moist corn-meal, with the production of ptomaines.

The *symptoms* at the beginning are languor, debility, indigestion, anorexia, restlessness, and occasionally diarrhea. This is soon followed by erythema, pain, and roughness of the skin. Exfoliation of the latter reveals a suppurating surface. In severe cases, paresthesiæ, spasms, paraplegia, headache, backache, delirium, and a suicidal mania may occur. Idiocy and profound cachexia may result from numerous attacks.

Structural changes have been found in the cord, and fatty degeneration and ulceration in the viscera.

Prophylaxis by thorough drying and careful storing of the meal is to be aimed at. The symptoms are to be met as rationally as possible.

**Lathyrismus** is an intoxication caused by the seed (used in the form of meal) of three varieties of vetch or chicken-pea, viz. *Lathyrus cicera*, *L. sativus*, and *L. clymenum*, or, respectively, red, German, and Spanish vetch. The meal is generally mixed with that obtained from other cereals. Its use for several hundred years has been observed to cause leg-stiffness, passing into a transverse myelitis, with sensory and motor paraplegia. Spasticity and exaggerated tendon-reflexes may remain for some time after the paralysis subsides. Slight fatty degeneration was noted by Cautain in excised bits of muscle. Very chronic cases may die in paralysis, from the toxic effects of the poison, which, thus far, has not been separated.

**Mushroom-poisoning.**—Though not so common as formerly, poisoning from eating non-edible mushrooms occurs now and then, owing to ignorance or carelessness in gathering, keeping, and cooking them. *Fresh morels* are poisonous, while those that have been dried and boiled are not so, because of evaporation or solution of the contained poison.

The *red agaric* (*amanita muscaria*), on account of the poisonous alkaloid muscarin that it contains, may cause very severe symptoms. These are nausea, vomiting, diarrhea, hemoglobinemia, hemoglobinuria, and jaundice (*probably hepatogenous*) in the case of fresh morel-poisoning (Strümpell). Tetanic and epileptiform convulsions give a slow pulse, dilated pupil, disturbed vision, salivation, coma, and death in the gravest cases of red-agaric intoxication, in addition to the symptoms of gastrointestinal irritation.

The *treatment* is symptomatic. Emetics, purgatives, stimulants, and, in red-agaric poisoning, atropin, for its physiologic antidotal effect, are usually indicated.

## OBESITY.

(*Polysarcia Adiposa*; *Lipomatosis Universalis*.)

**Definition.**—Corpulence, or the presence of an excessive amount of bodily fat, may be said to begin to take the form of a disease when it

becomes an inconvenience or impairs the bodily functions. Obesity is essentially a disease of nutrition.

**Pathology.**—The chief alteration is the marked and, in some instances, colossal increase in the fat deposit throughout the body. Not only is the adipose tissue greatly increased in localities where it is normally found or “preformed,” as under the skin, but the various internal organs and tissues that are normally quite or nearly free from fat, may in obesity show a decided fatty infiltration. Toldt affirms that in the graver cases of corpulence, in which marked depositions of fat are found in the viscera around and between the parenchymatous elements, the fat-cells are nothing more than transformed connective-tissue cells.

The round, fat face, “double chin,” broad and deep chest, large waist, thick and prominent, sometimes overhanging, abdominal *panniculus adiposus*, and bulky, cylindric, and apparently shortened extremities, are familiar appearances *postmortem* as well as *antemortem*.

There may be differences in the number and size of the fat-globules in the histologic elements. Thus, in the plethoric form of obesity the cellular fat-globules are larger than those of the anemic or hydremic form. Qualitative differences in the fat may also occur.

The *blood* in cases of obesity is increased in specific gravity to as much as 1065 or 1070. In a majority of cases the hemoglobin-percentage is also increased (plethora).

The *heart* is overlaid with fat, and the intermuscular tissue shows a decided fatty infiltration. Hypertrophic dilatation is frequently present.

The *arteries* may show fatty changes in the intima and media, and in the older cases chronic endarteritis with thickening and sclerosis of the vessels. The *veins* are often affected with varicosities.

Passive congestion and edema of the *lungs* are secondary to the cardiac weakness that is so common in advanced cases. For the same reason the *liver* and *kidneys* may be enlarged. Fatty infiltration may also affect and cause enlargement of the liver, and chronic interstitial nephritis may form a late complication of obesity.

The *stomach* may be dilated, and often shows a catarrh of the mucosa. Catarrhal enteritis of mild type also occurs sometimes. The pathogenesis of obesity has not as yet been fully determined.

**Etiology.**—Among the conditions *predisposing* to corpulence, the chief are heredity, climate, habit, occupation, temperament, age, and sex. In about one-half of the cases of obesity the tendency is inherited, and in these cases the abnormal increase of fat manifests itself early in life. Corpulence is much more frequent among the inhabitants of hot, moist climates, and of low countries of the temperate and arctic regions. Thus, it is commonly observed among Orientals, Dutchmen, South Pacific Islanders, Southern Italians, and certain African races. Sedentary habits and occupations form common predisposing factors. The sluggish, luxury- and rest-loving, phlegmatic temperament also favors an abnormal fat-deposition. As regards the age, polysarcia generally makes its appearance in persons of advanced middle life, between forty and fifty years, while congenital obesity is seen in infancy and early childhood; in women, it may appear at puberty and between thirty and forty years of age. Women, and especially Jewesses, seem to be more subject to corpulence than men. Congenital anomalies and monstrosities (idiots, cretins,



acephali), also anemics and hemiplegics, are often excessively fat. The prolonged use of arsenic may sometimes lead to fat-increase.

The *exciting* causes of obesity are especially the ingestion of too much fat-making food, the intemperate use of alcoholic beverages, with or without deficient exercise. The ingestion of food in excess of the bodily requirement of proteids, fat, and carbohydrates (which varies in different individuals), combined with insufficient assimilation or physical exercise, will result in an abnormal accumulation of fat. This is especially the case where a predisposition exists. The fat may be derived from the excess of albumin, fat, or carbohydrates in excess. Alcohol, especially in the form of beer, ale, porter, and the like, promotes fatty infiltration and degeneration. An excessive diet of starches and sugars acts indirectly as a fat-producer by lessening the oxidation of the ingested fat and of the fat formed from proteids, because the carbohydrates themselves are so readily oxidized.

**Symptoms.**—Obesity is not accompanied by any bodily symptoms at first. Except some inconvenience, and a sense of burdensomeness during walking or working, nothing may be complained of for years. With the progressive development of the disease, however, and particularly with the involvement of the viscera, subjective manifestations increase in number and intensity, and objective symptoms and physical signs also become more numerous and more marked. Usually the earliest troublesome symptom is breathlessness on exertion, due to a weak heart and to the hampering of respiration by heavy chest-walls and the upward-crowded diaphragm. In plethoric individuals the face is red and congested, as are also the mucous membranes (conjunctivæ, labiæ). In anemic subjects (usually women) the skin is pale, the muscles are flabby and weak; the pulse is small and compressible, and dyspnea, palpitation, inclination to rest often and sleep much, and dizziness (symptoms of anemia and chlorosis) are manifested. On the other hand, in plethoric, corpulent subjects (usually men), the muscles are firm and strong, and the pulse- and heart-beats vigorous; later, however, the latter become weak. Brachycardia is not infrequent, and the pulse-rate may become as low as 50 beats per minute. The signs of fatty heart (*vide* p. 656) are obtained on physical examination. Muscular power may diminish very rapidly, the appetite often fails, and, oddly enough, great, fat men may consume very small quantities of food. Intercurrent acute febrile affections (typhoid fever, pneumonia) are badly borne, and hyperpyrexia is usually associated with them.

The *liver* may show enlargement on percussion and palpation. The passive congestion of the *respiratory mucous membrane* is often signalled by cough and by an increase of the dyspnea. Profuse sweating is common. There may be *polyuria* or *oliguria*, according to the activity of the skin and kidneys at the same time. Uric acid and the urates are usually found to be increased.

Symptoms of *gastric catarrh* and *gastrectasia* may occur. Great thirst and bulimia are noted in some instances. Constipation may be followed by chronic diarrhea. Sexual desire is often abated, and azo-spermia is not rare. Corpulent women often suffer from uterine displacement and prolapse. Amenorrhea, sterility, endometritis (congestive), leukorrhea, and an aggravated climacteric are seen in obese women also.

The skin is often irritated (intertrigo) by the excessive sweating, and by the friction of cutaneous surfaces in the folds of fat, as under the breast and axillæ, at the navel, at the abdominal and inguinal folds, and around the scrotum and labia. Painful excoriations, pruritus, furunculosis, acne rosacea (in alcoholics), and alopecia are not uncommon.

**Complications.**—Hernia, cardiac asthma, bronchitis, pulmonary congestion, edema, arteriosclerosis, albuminuria, glycosuria, anginal attacks, Cheyne-Stokes respiration, cerebral hemorrhage, and coma may manifest themselves as the precursors of the final stage.

**Diagnosis.**—This is not difficult in most cases. Care and watchfulness must be exercised in detecting associated conditions, complications, and sequelæ.

The **prognosis** will depend upon the peculiar features of each individual case, the cause and its removability, and upon the variety, degree, symptoms, and prevailing complications.

**Treatment.**—**Prophylaxis** is important in the earlier years of those showing an hereditary predisposition to corpulence. The fat-forming (farinaceous) substances must be diminished in the dietary. The proportions of fat and proteid in the food must be regulated according to the amount of muscular activity, and the latter should be encouraged in fresh air, along with cool bathing. At middle life, in those predisposed to polysarcia, all imprudences in eating and drinking should be cautioned against, and the quantities of various articles of food and the time of eating regulated. Outdoor sports and gymnastics should be also gauged accordingly.

The **dietetic** treatment of confirmed obesity is all-important. The ingestion of foods that lead to the formation of fat must be limited. Inseparable from this is the stimulation of the bodily forces that oxidize and destroy the fat. These two means are utilized in the principal methods of treating obesity, and that method must be selected which invigorates, while at the same time it involves neither injury nor weakening of the patient.

The principal systems of dietary are those known by the names of Banting, Ebstein, and Oertel. In all of them the total amount of food is gradually diminished as long as there is an increase of the body-weight or a continuance of the subjective distress.

In "Bantingism," sugars, fats, and starches are greatly reduced in the diet-list; water, however, is not restricted, and vinous and spirituous liquors are rather freely permitted. In those of a lithemic, rheumatic, or gouty diathesis (often associated with obesity) Banting's heavy proteid and alcohol dietary is not to be recommended. It is best, I think, to exclude alcohol in most cases, owing to its effect in diminishing tissue-oxidation and in retarding cell-metabolism.

In Ebstein's diet-list more than double the amount of fat and carbohydrates is permitted as compared with Banting's list, whilst the albuminous substances are diminished. Fat is freely allowed, while sugar and potatoes only are strictly forbidden.

Oertel of Munich also allows more fat than Banting, but less fat and more (about double the quantity) proteids and carbohydrates than Ebstein. The amount of *free* water permitted daily is only one pint; about one pint additional in other food is allowable.

Oertel<sup>1</sup> writes: "The body stores up fat if more than 118 grams of albumin and 259 grams of fat, a total of 377 grams (2894 calories), are taken in. On the other hand, 110 grams of albumin and 600 grams of starch, a total of 710 grams (2944 calories), may be given without producing a deposit of fat. With a mixed diet the limit lies near 118 grams of albumin, 100 grams of fat, and 368 grams of starch, a total of 586 grams (2923 calories). If we want to bring about the decomposition of the fat already accumulated in the body, we do so best by diminishing the supply of fat and by permitting a certain quantity of carbohydrates." His diet-table for obesity is appended:

	Albumin.	Fat.	Carbohydrates.	Calories.
Minimum . . . . .	156	25	75	1180
Maximum . . . . .	170	45	120	1608

For fuller details, Oertel's tables,<sup>2</sup> giving a special diet-list in circulatory disturbances, may be consulted.

On the basis of Voit's laws, Strümpell recommends in the average cases 125 gm. (4 oz.) or more of albumin, 40 gm. (1 $\frac{1}{4}$  oz.) of fat, and 150 gm. (4.62 oz.) of starch. Schwenniger's rule differs from Oertel's merely in the forbidding of liquids with the meals and in permitting their use only after two hours have elapsed. Yeo's diet-list is also a useful guide.

Under any system of dietetic treatment the patient should be weighed accurately and frequently, and the food-limit be diminished or modified according to the results. The food may be weighed and measured at first, but the patient soon learns to estimate by bulk the requisite quantity of each substance.

The following dietary illustrates what may be ordered in some cases of obesity:

*Morning Meal.*—Fine wheat-bread, 1 $\frac{1}{4}$  ounces (40.0); a soft-boiled egg; milk, 1 ounce (32.0); sugar, 77 grains (4.9); coffee, 4 $\frac{1}{4}$  ounces (136.0).

*Noon Meal.*—Soup, 3 ounces (96.0); fish, 3 ounces (96.0); roast or boiled beef, veal, or game or poultry, 6 to 8 ounces (192.0–256.0); green vegetables, 1 $\frac{1}{2}$  ounces (48.0); bread, 1 ounce (32.0); fruit, 3 or 4 ounces (96.0–128.0); no liquid (or only 4 or 5 ounces—120.0–148.0 cc.—of very light wine).

*Afternoon Meal.*—Sugar, 77 grains (4.9); coffee, 4 ounces (128.0); milk, 1 ounce (32.0); occasionally bread, 1 ounce (32.0).

*Evening Meal.*—Caviare,  $\frac{1}{3}$  ounce (10.6); one or two soft-boiled eggs; beefsteak, fowl, or game, 5 ounces (160.0); salad, 1 ounce (32.0); cheese, 1 dram (4.0); bread, rye or bran,  $\frac{1}{2}$  ounce (16.0); fruit or water, 4 to 5 ounces (120.0–148.0).

The **mechanical treatment** of corpulence, by exercise, is to be used in conjunction with the dietetic. The form of the exercise, and also the time and frequency, must be adjudged for each case. When cardiac dilatation and myocardial degeneration (fatty) are the cause of symptoms of precordial distress, dyspnea (however slight), and palpitation, resort may be had to Oertel's system of graduated walking on the level

<sup>1</sup> *Twentieth Cent. Pract. of Med.*, vol. ii. pp. 698, 699.

<sup>2</sup> *Loc. cit.*



or climbing along "health paths" (*vide* Fatty Overgrowth, p. 657). Or, the well-known Nauheim or Schott treatment may be used. Great care must be exercised in prescribing the mechanical treatment in obese persons who have atheromatous vessels.

The medicinal treatment is neither satisfactory nor successful. The juice of the phytolacca berry may reduce the weight, but it usually does so at the expense of bodily strength.

Recently, the use of thyroid extract has come into favor, and this, judiciously given, promises good results. Leichtenstern, Wendelstadt, Ewald, and others have reported success in a number of cases, especially in those exhibiting the anemic, flabby, "myxedematoid" form of obesity. The loss of weight was from 2 to 3 pounds (1-1.5 kgms.) in one week, and as high as 20 pounds in two to four weeks. In two of my own cases belonging to this category the use of thyroid extract (desiccated) in small doses (gr. j—0.0648, t. i. d.) caused a progressive loss of weight at the rate of 4 and 6 pounds per week respectively, without injury to the general health. Thyroidin, the active principle of the thyroid gland, as shown by Baumann and Ross, gives results that are perhaps as good as those of thyroid-feeding. Jcozykowski treated 10 cases of corpulence by thyroidin in doses from 5 to 8 grains (0.324-0.518) *per diem*. In 1 case more than 40 pounds (18.1 kgms.) were lost in two months, and in another 30 pounds (13.6 kgms.) in three months. Symptoms of thyroidism are the signal for a reduction in the dosage of thyroid extract (*vide* Myxedema, p. 464).

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## HEAT-STROKE.

(*Sunstroke; Insolation; Thermic Fever; Heat-exhaustion; Heat-prostration.*)

**Definition.**—A diseased condition the effect of exposure to excessive heat.

**Pathology.**—*Rigor mortis* is marked and comes on early. The high temperature of the cadaver accelerates the putrefactive changes, which also appear early. There is considerable venous engorgement of the brain and of the cerebral and spinal membranes; also of the lungs, spleen, and conjunctiva. The blood is fluid and dark, and the corpuscles are crenated and do not tend to form rouleaux. Ecchymoses and extravasations of blood are found in the skin, the serous membranes, and the cavities, around the superior (cervical) sympathetic ganglia and the vagus and phrenic nerves. Parenchymatous changes in the liver and kidneys may be found. Rigid contraction of the left ventricle is a notable feature, while the right ventricle is usually dilated with blood. Van Gieson's recent report of the cellular pathology of the cerebro-spinal system in 3 cases of sunstroke in New York shows an acute parenchymatous degeneration of the neurons of the whole neural axis similar to that of, and, Van Gieson thinks, here actually due to, "a species of auto-intoxication." He found the chromophilic plaques in the cortical cerebral and cerebellar (Purkinje's) cells and also in the cells of the anterior horns of the spinal cord, diminished in number, changed in shape and

position, sometimes finally broken up, and even entirely absent. The nuclei stain more deeply than normally.

**Etiology.**—Anything that lessens bodily resistance to external high heat predisposes to heat-stroke. Thus, privation, unsanitary surroundings, fatigue of body or mind, emotional excitement, worry, and excessive fretfulness, overeating, indulgence in alcoholics (especially), and previous attacks of sunstroke, are all conducive to heat-stroke on exposure to high temperature. Males are affected more often than females.

**Sunstroke** occurs in persons (on land) working hard under the direct rays of the sun, in an atmosphere that is very hot and humid, still, and sultry. Soldiers on the march and heavily accoutered, masons, bricklayers, hod-carriers, roofers, drivers, farmers, and other out-door laborers are particularly subject to insolation.

**Heat-stroke** and **thermic fever** are terms more appropriately applied to those similarly affected in midsummer while working in places not exposed to the sun, but yet close, confined, and excessively hot, such as glass-works, foundries, ocean steamers, stoke-holes, boiler-rooms, steam laundries, sugar-refineries, kitchens, and the like.

**Heat-exhaustion** (*prostratio thermica*) is caused under similar conditions as the preceding, but manifests dissimilar, and sometimes almost opposite, effects.

The majority of the cases of sunstroke occur between 2 and 5 P. M., although heat-stroke and heat-exhaustion may occur at night as late as 10 or 11 P. M., as among bakers, night engineers, and hotel cooks.

It seems to be the consensus of opinion that the direct cause of the symptoms of sunstroke, heat-stroke, or heat-prostration is the action of the excessive heat upon the heat-centers, or upon the vasomotor center or nerves (H. C. Wood), the former of which, if paralyzed, produces "thermic" or "heat-fever," while the latter, if paralyzed, produces *heat-exhaustion*.

It should be stated, however, that Lambert and Van Gieson,<sup>1</sup> after a clinical and pathologic study of 805 cases of sunstroke occurring in New York City during 1896, hold to the not improbable view that the immediate basis of sunstroke is autotoxic, with heat only as a contributing cause.

**Symptoms.**—Two forms of heat- or sunstroke are usually met with: (1) The *asphyxial* or *apoplectic* form; (2) the *hyperpyrexial* form. Flint believes that the majority of the cases of sunstroke are combinations of apoplexy and exhaustion. Vallin puts all cases of insolation into two classes: the first, sthenic or asphyxial, corresponding to our hyperpyrexial or congestive variety; the second, asthenic or syncopal, corresponding to our heat-exhaustion. Mixed forms may occur quite frequently, the most prominent symptoms being referable to the organs suffering the most, as the cerebro-spinal system, heart, lungs.

**Heat-apoplexy** (*asphyxial sunstroke*) is probably the least frequent form. There may be sudden premonitions, or dizziness, chromatopsia, throbbing headache, cessation of sweating, or dyspnea. Sometimes the patient, while at work in the sun, suddenly falls unconscious, a few convulsions may occur, and in this state he may die with symptoms of cardiac failure. More often, insensibility is not so profound as complete

<sup>1</sup> *Med. News*, July 24, 1897.

coma, there is much restlessness, epigastric "cramp" may be complained of, also a sense of thoracic oppression, and occasionally there are nausea and vomiting. The headache may be intense, the face is flushed, the pulse is rapid and full, the temporal and carotid arteries are bounding, the breathing may be labored and stertorous, the pupils are contracted (except in grave cases), and urination is often frequent. The skin is hot and dry, and may show petechiæ. The tongue is coated with a whitish fur. A wild delirium has been observed in some cases. The temperature may be subnormal, and is not higher than 102° F. (38.8° C.) in many instances. In others, a mild degree of thermic fever may be associated with the apoplectic condition, the thermometer registering 104°–106° F. (40°–41.1° C.). In fatal cases the coma becomes deeper and deeper, the pulse more rapid and feeble, and Cheyne-Stokes respiration may precede the termination. A "mousey" odor about the body has been noted. In favorable cases the temperature falls to normal by lysis in three or four days, consciousness being rapidly regained at the same time.

The *hyperpyrexial* variety comprises the numerous cases of marked sunstroke that resemble the preceding type, with the addition of an intensely high temperature (*thermic fever*). The patient may suddenly become comatose and die in an asphyxiated condition, with a temperature as high as 110°–115° F. (43.3°–46.1° C.) or even higher.

Sometimes prodromes, as anorexia, progressively increasing physical weakness, cramp-like abdominal pains, irritability and restlessness, vertigo, colored and blurred vision, lack of sweating,

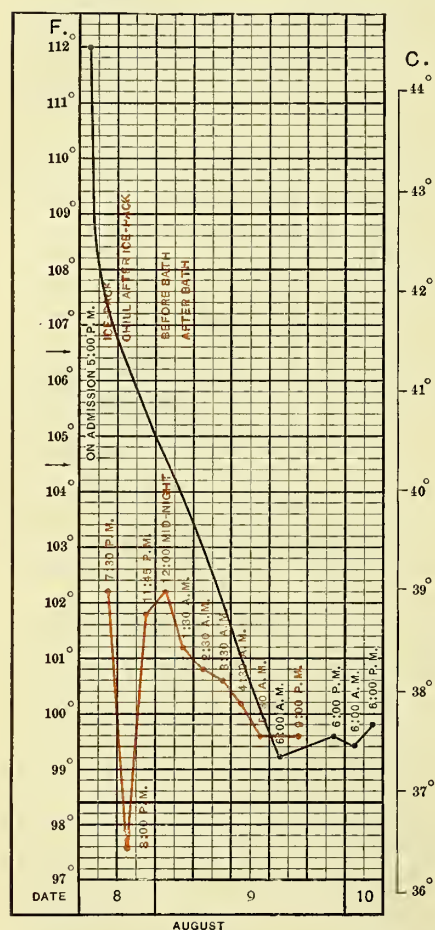


FIG. 79.—Chart of a case of sunstroke. J. D., aged forty years; steam-fitter. Recovery.

a "bursting" headache, and an irritable bladder may exist for several days. A subconscious (automatic) state, in which the patient may be unaware of his surroundings, although walking or even working, may be noted for hours before he is stricken down. The onset is marked by hyperpyrexia; the skin is hot, burning, dry, sometimes flushed and red, and sometimes cyanotic and clammy; the eyes are suffused or "staring and filling," with pin-point pupils. There is a full, rapid, and non-



compressible pulse, and coma may be present. Clonic spasms may alternate with either muscular rigidity or flaccidity. Delirium, moaning, jactitation, and explosive expiratory sounds may occur. There is frequently incontinence of both feces and urine. The temperature is very high in most of the cases, varying from  $105^{\circ}$  to  $112^{\circ}$  F. ( $40.5$ – $44.4^{\circ}$  C.).<sup>1</sup> The pulse-rate varies with the temperature, from 90 to 160 beats per minute. The respirations are also increased to 24–50 per minute. Many of the alarming symptoms, including the high fever (*vide* accompanying chart, Fig. 79), unconsciousness, cyanosis, dyspnea, and convulsions, may greatly subside during and after the use of the cold bath. Secondary exacerbations occur for a few days before convalescence is established in the favorable cases (*vide* chart, Fig. 80).

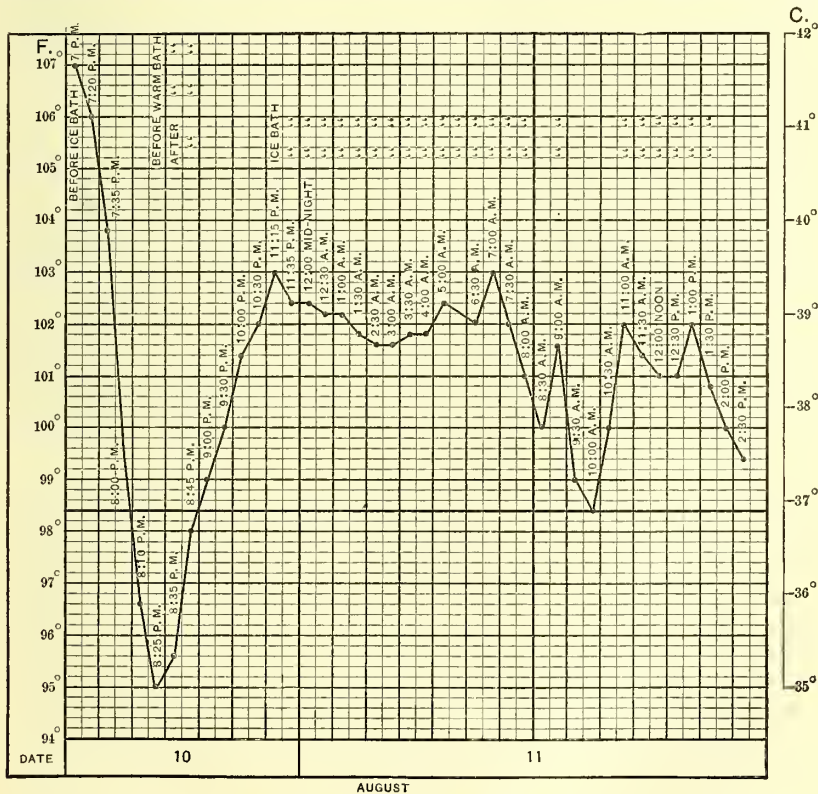


FIG. 80.—Chart of a case of sunstroke. C. B., aged twenty-nine years. Recovery.

Some patients never rally, and die in a state of asphyxia. Retention of urine (suppression) is observed at times, and particularly in those accustomed to the use of alcohol. Leukocytosis is noted, besides the crenation of the erythrocytes (degeneration of the red cells).

Fatal complications of sunstroke are pneumonia, meningitis, uremia, and cardio-respiratory paralysis.

Heat-prostration or heat-exhaustion may come on gradually or suddenly, with prodromal symptoms (dizziness, faintness, headache, nausea,

<sup>1</sup> Lambert (*loc. cit.*), reports a case in the N. Y. Hosp. of  $117.8^{\circ}$  F. ( $47.6^{\circ}$  C.).

thirst, drowsiness, yawning, epigastric or lumbar pains, numbness and tingling of the hands and feet). These are followed by coldness, clamminess, and pallor of the surface, marked muscular weakness and prostration, a small, febrile, rapid pulse, sighing breathing, syncope, and collapse in the graver cases. The temperature at first is subnormal ( $95^{\circ}$  to  $97^{\circ}$  F.— $35^{\circ}$  to  $36.1^{\circ}$  C.), though mild thermic fever of from  $100^{\circ}$  to  $102.5^{\circ}$  F. ( $37.7^{\circ}$ – $39.1^{\circ}$  C.) may be present. Consciousness is rarely completely absent and is regained early. Recovery usually takes place within one or two days, and in milder cases, under prompt and appropriate treatment the patient may be ready to go about in a few hours. In a few cases of extreme prostration in weakly persons death may ensue from cardiac failure.

The sequelæ of heat-stroke are quite interesting and peculiar in some instances. Osler relates the case of a patient who “was subsequently so sensitive to temperatures in the neighborhood of  $75^{\circ}$  F. ( $23.8^{\circ}$  C.) (*italics mine*) that at such times he lived comfortably only in the cellar, and finally sought refuge in Alaska.”

Chromatopsia, severe headaches, irritability and ugliness of temper, or delirium may occur in some patients as soon as warm weather sets in, and may be due occasionally to chronic meningitis (Wood).

**Diagnosis.**—Bearing in mind the characteristic differences that are outlined above between sunstroke (including the asphyxial and hyperpyrexial forms) and heat-exhaustion, the diagnosis is not difficult. The history and circumstances attending the seizure are also important in making the diagnosis. From other affections, as *acute alcoholism*, *meningitis*, *uremia*, and *cerebral apoplexy*, the differentiation is readily made by noting the previous history, mode of attack, presence or absence of thermic fever, state of consciousness, urine, skin, pupils, pulse, respiration, and nervo-muscular apparatus.

**Prognosis.**—This is usually favorable in cases of heat-prostration. It is less so in sunstroke, but in all cases it depends on the severity of the stroke, the previous health and habits of the patient, the complications, and the promptness and facility of the treatment. The mortality-rate during a prolonged period of excessively hot and humid weather may be very high, ranging from 15 to 50 per cent. In New York City, during the week ending August 15, 1896, out of a total number of 1810 deaths, 648 were reported as due to sunstroke (Lambert).<sup>1</sup>

**Treatment.**—**Prophylaxis.**—This is highly imperative in hot, sultry weather, particularly in cities, in which persons must work in the sun or in poorly-ventilated and highly-heated, closed places. Workmen should be taught and warned privately and publicly, as through the medium of the press and Health Board circulars, to take extra precautions during hot weather, to work and sleep in as well-ventilated rooms as possible, and to secure artificial ventilation, if necessary. They should live regular and temperate lives, avoiding alcohol and heavy eating; oat-meal water should be drunk, light-weight and light-colored clothing should be worn, and the direct rays of the sun should be avoided as much as possible. The condition of the skin should be watched and care taken that sweating continues freely. Shelter or rest should be sought at once if sweating stops. Cool wet cloths or green

<sup>1</sup> *Loc. cit.*

leaves should be worn inside a light straw hat, and sometimes it may be necessary for employers to shorten the hours of labor during the hottest part of the day.

**Treatment of the Attack.**—Cases of ordinary *heat-prostration* seldom require much treatment beyond the removal of the patient to the shade of a comparatively cool place, loosening all constricting clothing, spraying with cool water, the use of ammonia- or amyl-nitrite-inhalations, and of the aromatic spirits of ammonia or spiritus glonoīni by the mouth. If the temperature is subnormal and collapse threatens, a hot bath is advisable. Strychnin and digitalis may be used for a day or two to combat the nervo-muscular weakness.

*Heat-stroke*, especially the hyperpyrexial cases, must be promptly treated by the application of the ice-bath (ice floating in a tub of water), temperature about 40° F. (4.4° C.), or by rubbing, by the cold pack, or by the needle-spray with iced water.

In the *asphyxial* cases venesection is frequently indicated. External stimulation should be applied to the precordium by mustard and to the feet by hot bottles, and hypodermic injections of nitroglycerin, strychnin, atropin, brandy, camphor, or ether are useful. Ice should be rubbed over the head constantly. Care should, however, be taken to see that the temperature is not reduced too far. A temperature of about 102° F. (38.8° C.) should be the signal for cessation of the ice-bath, and for the removal of the patient to a cot, where he is to be rubbed dry and allowed to rest until an exacerbation of fever indicates the reapplication of the cooling measures. Ice-water enemata, with or without brandy, are often useful adjuvants. The needle-spray of cold water is an excellent nervous stimulant as well as antipyretic. It is given while the patient lies on a Kibbee or netting cot, or on a cot covered with a rubber sheet so arranged as to drain into a pail or trough. Internal antipyretics are seldom well absorbed, and their depressant action is so well known as to discourage their use in place of hydrotherapy. Hutchinson, Coplin, and Bevan recommend highly the use of morphin to control the convulsions of heat-stroke. Artificial respiration in the asphyxial cases, kept up until other measures and stimulants have time to act, may be the means of saving life.

After the reduction of the hyperpyrexia the patient should be lightly covered on a cot placed in a cool place. An ice-cap should be applied to his head, and small pieces of cracked ice may be given to allay gastric irritability, with calomel to open the bowels if necessary. Albumin-water, skimmed milk, buttermilk, unfermented grape-juice, junket, and the like may be given for several days preparatory to the ingestion of heavier food. If, as sometimes happens, free diaphoresis does not come on after the reduction of most of the fever and the stimulating treatment, a hot bath may be given, and perhaps aided by the hypodermic injection of pilocarpin in urgent cases. *Sequele* must be treated on general principles.

The increased susceptibility to repeated attacks of insolation (after the first attack) makes it necessary to avoid exposure to heat ever after, and, if possible, to seek a cooler climate during the hot months.



## PART XI.

# ANIMAL PARASITIC DISEASES.

### PSOROSPERMIASIS.

*Psorosperms* belong to the lowest form of protozoa. They are also known as *sporozoa*, and, because of their parasitic relation to cells, as *cytozoa*.

The *amœba coli* of amebic dysentery belongs to the protozoa. Blood parasites (*hematozoa*), as the *plasmodium malaria*, are likewise closely related to the sporozoa.

Various coccidia may occur in man to produce the disease indicated by this heading. The *coccidium oviforme* of the rabbit is the commonest variety, being found also in rats and mice. It escapes from the livers of the latter animals and passes into the dejecta; it produces an hepatic disease in which there are numerous whitish nodules studding the liver. These range in size from a pinhead to a split pea, and on section disclose a bile-duct, the dilated portion of which forms the nodule. The ovoid coccidia are found in the epithelial cells of the walls of these biliary expansions.

The *coccidium perforans* and *coccidium bigeminum* are found in the cells of the intestinal villi instead of in the liver of the hosts mentioned above. Among veterinarians a common form of sickle-shaped organism is known that is found within an ovoid body in the sarcolemma of the pig's muscle—(*i. e.* the so-called Rainey's tube).

In man, hepatic disease similar to that found in the rabbit is produced by the *coccidium oviforme*. The tumors formed by the coccidia may be palpable, and the liver may be quite tender. Some chilliness and fever, malaise, and stupor passing into coma have been observed. Death was caused on the fourteenth day in a case admitted to St. Thomas's Hospital (Osler). The necropsy showed whitish neoplasms in the peritoneum, omentum, and kidneys.

In the intestinal variety of *internal psorospermiasis* nausea and vomiting, diarrhea, and the typhoid state may be manifested. Involvement of the kidneys has caused hematuria and frequency of urination.

*External* or *cutaneous psorospermiasis*, one form of which was formerly called *keratosis follicularis*, is characterized by lesions at first of a hard, crusty, papular type, later becoming confluent, and situated on the face, lumbo-abdominal, and inguinal regions. These papillomatous growths contain numerous parasitic sporozoa.

In carcinoma, epithelioma, and Paget's disease of the nipple coccidia are readily found in and between the pathologic epithelial cells,

but whether they have an etiologic bearing upon these malignant affections is still a matter of uncertainty.

*Prophylaxis* consists in cleanliness and care in preparing such food vegetables as spinach, lettuce, cabbage, and other greens that may possibly be contaminated by the excreta of the lower animals liable to psorosperm-infection. The **treatment** of psorospermiasis is symptomatic, though rectal injections of a solution of quinin (1 : 5000 to 1 : 1000) may be tried.

## DISTOMIASIS.

(*Trematodiasis*.)

VARIOUS forms of trematodes, including the distomata, may become parasitic in man.

**Distoma Hepaticum** (Liver-fluke).—Among the more common varieties of trematodes or flukes, is the distoma hepaticum or liver-fluke, a parasite found in animals (horse, goat, ass, sheep, rabbit) and accidentally ingested by man.

It is almost 30 millimeters (1.1 inches) in length, and inhabits the biliary passages of the animal, and from them is discharged into the intestinal tract and evacuated with the feces. Under certain conditions of temperature and moisture, a ciliated embryo escapes from the egg, and is ingested by a gasteropod or snail (*Limnaea truncatula*), in which it undergoes development into a sporocyst, that in turn gives origin to *radie* or parent nurses. These give birth to daughter-radie or *cercarie*, which leave the gasteropod or snail and attach themselves to aquatic plants, where they are in turn eaten by animals.

**Symptoms.**—When present in sufficient numbers in the bile-passages the liver becomes greatly enlarged, with the occurrence of jaundice and ascites that may prove fatal. Other symptoms may also be present; thus pain was prominent in 41 out of 100 cases reported by Kurimato in Japan, and heart-murmurs were present in 42 of those cases.

Late in the disease the liver may become nodulated and terminate in atrophy.

On inspection in well-marked cases, a peculiar barrel-shaped bulging is sometimes seen, extending over the hepatic area, with tense abdominal walls over the enlarged liver. This is a pathognomonic symptom of hepatic distoma. An endemic form occurring in Japan has been described; it is characterized by marked emaciation, diarrhea, hepatic enlargement, and often by ascites.

The **prognosis** of distoma hepaticum is absolutely fatal and the **treatment** is merely palliative.

Among other trematodes may be mentioned (a) *distoma lanceolatum* (found also in cattle); (b) *distoma crassum*, which is larger in size than the preceding; (c) *distoma sibiricum*; (d) *distoma pulmonale* (*D. Ringeri*); (e) *distoma spatulatum* (endemicum); (f) *amphistomum hominis*; (g) *distoma hematobium* (Bilharz). Two of these deserve extra, though brief, mention.

**Distoma Pulmonale** (*D. Ringeri*) (*Bronchial-fluke*; *Parasitic Hemoptysis*).—This parasite is very common in Japan. It finds lodgement primarily in the lung, and its ova sometimes form emboli in the brain, liver, and other tissues, and may also be found in the form of little cysts throughout the body. The symptoms are a cough, a reddish-brown bloody sputum, and the presence of the flukes in the expectoration. The latter are club-shaped, and are about 8–10 mm. ( $\frac{1}{3}$  in.) long.

**Distoma Hematobium** (*Bilharzia hematobia*; *Blood-flukes*).—This hematode is a narrow worm with anterior abdominal sucking-disks. The male is shorter and thicker than the female; the former being 4–15 mm. ( $\frac{1}{6}$ – $\frac{3}{8}$  in.) long; the latter, about 20 mm. ( $\frac{4}{5}$  in.). It prevails mostly in Egypt, Cape Colony, and other parts of Africa, and its entrance into the human body is now believed to be through the skin of those who bathe frequently in the African rivers, in many of which it abounds. It is not unlikely that, as formerly held, infection may also occur in many cases from drinking the impure water of the rivers. The parasites or their ova are found in the bladder, the pelvis of the kidney, and the veins (especially the portal and mesenteric).

The **symptoms** are hematuria, with some pain during urination. Pus, and some of the ova of the parasites, may also be found in the urine. No serious systemic disturbances occur in bilharziosis. Prophylaxis as regards drinking and bathing in African waters should be exercised. Fouquet affirms the value of the extract of male-fern internally in this form of distomiasis.

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## NEMATODES.

HELMINTHOLOGISTS include in this class the cylindric worms, certain varieties of which are among the most common entozoa that infest the human body and inhabit the intestines.

### ASCARIASIS.

**Ascaris Lumbricoides** (*Round-worm*).—**Natural History.**—This species resembles the common earth-worm, and is the most frequent in occurrence of all the parasites. It usually appears in children between the ages of three and ten years. The round-worm inhabits the upper portion of the small intestine, and occurs singly or in numbers. Its body is round, fusiform, and marked with fine transverse striæ. It has a yellowish or reddish-brown color, and measures in the female from 7 to 14 inches in length (17.5–35 cm.), and from 4 to 8 inches in the male (about 20 cm.), its thickness being about that of an ordinary goose-quill. The cephalic extremity of the worm has three oval papillæ, furnished with fine teeth; the caudal extremity is straight in the female and curved in the male.

Lumbricoid worms develop from ova, which are about .05 to .06 mm. long, elliptic, dark-reddish in color, and have a thick, resisting envelope. There may be sixty million of them in a single female worm, and they sometimes occur in the feces in vast numbers. The development



of the embryo and worm external to the body is not accurately known. The eggs obtain entrance into the human intestine most probably through drinking-water, and it has been held that abundant mucus, and the predominating starchy and saccharine diet of which children so often partake, offer a favorable nidus for the development of the ingested ascaridian eggs.

The round-worm sometimes, though rarely, migrates from the small intestine. It has been vomited up, and it has also crawled into the pharynx, mouth, and nares, and has been withdrawn thence by the patient's fingers. It has even passed into the larynx and trachea, causing fatal asphyxia or pulmonary gangrene. The Eustachian tube and biliary ducts may be invaded with such serious symptoms as perforation of the membrum tympani and hepatic abscess. The ascarides have also been found in the peritoneal cavity, *postmortem*, with intestinal perforation, due, most likely, to other causes. They may penetrate the pancreatic duct and enter fistulæ connected with the intestine.

**Symptoms** may be absent, and yet the worms be found repeatedly in the stools. Existing symptoms are indefinite, and point simply to an irritative condition of the bowel. Serious symptoms may, however, result from the migration of the worm, as into the biliary passages, Eustachian tube, or larynx. Fever is not a necessary concomitant. Lumbricoid worms may give rise to any or all of the following symptoms: colicky pains, nausea, vomiting, indigestion, diarrhea (sometimes), restlessness, irritability, anorexia, itching of and picking at the nose, disturbed sleep with grinding of the teeth, salivation, and nervous twitchings. The child's abdomen and face may be swollen. Very nervous children may manifest epileptiform convulsions, choreic movements, dilated pupils, vertigo, cephalalgia, mental disturbances, and even contractures.

**Complications.**—The development of jaundice will indicate obstruction of the bile-duct, in cases in which the worms have been found in the feces. So also, suffocative symptoms coming on, especially at night, in a child with worms, may be due to a migrating lumbricoid. Perineal abscesses and inflamed herniæ that have perforated externally sometimes discharge the ascaris lumbricoides.

**Diagnosis.**—This is positively determined only by discovering the worm or ova in the stools. In doubtful cases, judged symptomatically, the administration of a suitable purgative and inspection of the resultant passages will enable the physician to arrive at a diagnosis.

The **prognosis** is good, unless serious complications arise (*vide supra*), when the case should be guarded accordingly.

**Treatment.**—*Prophylaxis.*—The water used for drinking-purposes should be obtained from the purest sources. That from small streams, shallow wells, and the like is most likely to contain the ova of the lumbricoides, and should be avoided. The use of filtered water should be encouraged.

Before giving an anthelmintic, it should be borne in mind that no good result can be certainly obtained unless the gastro-intestinal tract be nearly deprived of food for from twelve to thirty-six hours, so that the toxic action of the drug used may be exerted directly upon the unprotected worm.

Santonin is at once the most efficient and the most easily administered remedy. It may be given in doses of gr.  $\frac{1}{4}$  to 1 (0.0162–0.0648) of the crystals to a child, or from gr. ij to iv (0.1296–0.2592) to an adult, in the form of a troche, before breakfast. A little milk or other light nourishment may be allowed, the troches being continued once or twice daily for two or three days. This treatment is to be followed by a brisk purge, preferably gr. j to iij (0.0648–0.1944) of calomel. I have sometimes combined small doses of calomel with the santonin in a troche, and with good effect. Xanthopsia or yellow vision, spasms, and even convulsions, and saffron-colored urine may follow the use of santonin in cases of idiosyncrasy or overdose of the drug. Oil of worm-seed (*chenopodium*) in doses of five to ten drops, in emulsion, capsules, or on sugar, may also be used with benefit. Another favorite remedy with some is the unofficial fluid extract of *spigelia* and *senna*, to be given in from 1- to 3-dram (4.0–12.0) doses. Finally, the fluid extract of *spigelia* alone (1 to 2 drams—4.0–8.0), followed by a brisk purge, may bring away dead worms.

**Oxyuris Vermicularis** (*Seat-, Pin-, Thread-, or Maw-worm*).—**Natural History.**—The *ascaris vermicularis*, as this worm is also called, inhabits the colon and especially the rectum. It is a small worm, as several of the commonly-used terms signify, and frequently it occurs in great numbers, sometimes agglutinated with mucus into feculent balls. It is most common in children, though found not rarely at any period of life. The female oxyuris is whitish in color and about ten or twelve millimeters (one-half inch) long, the male being about three or four millimeters (about one-sixth of an inch) in length. Oxyures develop from ova in about two weeks after the ingestion of the latter. The eggs are irregularly ovoid, about  $\frac{1}{500}$  in. (0.05 mm.) in length, and tenacious of life. By the time the embryos have reached the cecum, they are sexually mature, and when the female arrives in the rectum, immense numbers of eggs are deposited that mature into great numbers of worms, the latter being discharged with the feces. Sometimes the worms crawl out of the anus.

Infection with the ova may take place through water and food (green, uncooked vegetables and fruit) that have come in contact with the hands of infected persons. Scratching the anus will permit of the reception of oxyuris eggs under the finger-nails (Zenker and Heller), and in careless, ignorant, and uncleanly persons the possibility of such an auto- or re-infection should be recognized and avoided.

**Symptoms.**—*Pruritus ani* (itching of the anus), sometimes burning pain, and tenesmus, with restlessness and disturbed sleep, are the commonest symptoms of the presence of this parasite. The itching is always worse at night, and may be paroxysmal. An herpetic or eczematous eruption around the anus should arouse suspicion, particularly in children, of the presence of the oxyuris in the rectum, and it accounts for the intense itching (Flint). Anorexia and anemia, rectal irritability, and “nervousness” may be associated. It is believed that the migration of the worms into the vagina of girls may set up pruritus and leukorrhea, and that habits of masturbation may be induced in both girls and boys by the sexual irritation caused by the worm. Inspection of the stools will reveal, in positive cases, the whitish, thread-like parasites.

**Diagnosis.**—The pruritus, indicating rectal trouble, will direct the physician's attention to the anus, where the oxyures may be seen; if not found, their discovery in the feces or the discovery of the eggs by microscopic examination will suffice.

The **prognosis** is good, and proper treatment is always effective.

**Treatment.**—The exhibition of anthelmintics and purgatives, such as recommended for destroying and removing the lumbricoid worm, may be effective against seat-worms also, but mainly in reaching those lodged in the bowel above the rectum. Attacking the oxyures directly, however, by means of enemata is the most useful and rational treatment.

The rectum should be well emptied of feces, so that the worms may be exposed to the action of the medicament injected, and for this purpose enemata of cold water, either simple or with salt or soap, may be resorted to. Injections containing the decoction of quassia (1 or 2 ounces—32.0 to 64.0—of the powder or chips to the pint—half liter—of water) are nearly always curative. Other useful remedies are carbolic acid, turpentine, tannin, vinegar, camphor, potassium sulphid, and the oil of eucalyptus. The injections should be repeated once or twice daily for at least ten days. It sometimes happens that killing the worms as directed above affords only temporary relief. The reason for this is obviously to be found in the fact that the oxyuris breeds in the cecum, and that only grown forms descend, reaching the rectum.

Rectal irritation may be allayed by injections of laudanum and starch-water (gtt. iij—v to the ounce—32.0). Anal itching is often amenable to carbolized vaselin, applied at bed-time, or to belladonna ointment, or the following, which has been highly recommended:

R <sub>y</sub> . Hydrarg. chloridi mitis,	ʒij (2.592);
Petrolati,	ʒss (16.0).

M. et ft. ung.

Sig.—Apply at bedtime.

**Ascaris Alata.**—This is another name for the *ascaris mystax*, a species of worm found in the intestines of the dog and cat, and occasionally in man. It is a slender worm, with a closely-rolled spiral tail and a wing-like projection on either side of the head. The female is about 6–7 centimeters (2.7 inches), the male about 4 centimeters (1.75 in.) in length. Scarcely ten instances, however, have been recorded in which this parasite has occurred in man.

**Trichocephalus Dispar** (*Ascaris trichiura*).—**Natural History.**—This worm measures about four or five centimeters (2 inches) in length, and is characterized by the very slender, hair-like appearance of the anterior two-thirds of its body, in contrast to the thick posterior portion, which is more or less straight and blunt-pointed in the female, but rolled into a spiral in the male. Its particular habitat seems to be the cecum, though sometimes it is also found in the colon. It may exist in great numbers. Europeans appear to be infected with the parasite more commonly than Americans. The trichocephalus has been found *postmortem* in many subjects dying with various diseases, as typhoid fever (Flint), meningitis (Barth), profound anemia (Osler), and beri-beri.

Propagation is effected by the microscopic eggs, which are ovoid, hard, nodular, brownish, and about 0.05 mm. ( $\frac{1}{200}$  in.) in length.



**Symptoms.**—It is not certain that the parasite causes any symptoms, nor even that it aggravates those of an associated disease (*vide supra*). When occurring in great numbers the possibility of fecal accumulation may be mentioned.

The **diagnosis** may be made by microscopy. The ova may be detected in the feces.

The **prognosis** and **treatment** are not called for.

#### ANKYLOSTOMIASIS.

**Ankylostomum Duodenale** (*Dochmius duodenalis*).—**Natural History.**—This parasite belongs to the family of *strongylidae* of the nematoid worms. It was discovered in Milan, in 1838, by Dubini. The length of the female is from 8 to 18 mm. ( $\frac{1}{2}$  inch), and of the male from 6 to 10 mm. ( $\frac{1}{3}$  inch). Its body is thread-like, with a conical-shaped head, and a large, bell-shaped mouth surrounded by a horny capsule, and possessing four hook-like teeth, ventrally situated, and two smaller, vertical teeth on the dorsal side, by which the worm fixes itself to the mucous membrane. A bulbous-like swelling exists at the tail end of the male worm. It inhabits the jejunum and duodenum. The eggs are found in muddy water, and there liberate the embryos. These develop into larvæ, which, when taken into the human bowel through drinking-water develop into mature worms. They do not multiply within the intestine.

**Pathology.**—The ankylostomum is nourished by the blood it sucks from the intestinal vessels. It is found *postmortem*, sometimes, in the mucous or even submucous coat, rolled up in a little blood-cavity. Echinomoses, containing a central opening through which blood can ooze, are the usual result of the worm's action. Chronic catarrhal enteritis is usually associated. Hypertrophic dilatation of the heart is observed.

**Symptoms.**—The chief symptom of the condition is anemia (secondary). When the number of ankylostoma embryos introduced into the intestine is large, the anemia may develop acutely; when but a few are introduced, the withdrawal of blood is more gradual, and chronic anemia develops. I think, however, it may be safely affirmed that the anemia is not wholly due to blood-sucking. In some cases the impoverishment of the blood has been so profound as to simulate a pernicious anemia.

This parasite has been found to be the cause of the disease known as "Egyptian chlorosis," first described by Griesinger. Ankylostomiasis is not uncommon in tropical countries (Italy, Brazil). In Italy it has been termed *tunnel* or *mountain anemia*; in Belgium it is known as *brickmaker's anemia*; again, it occurs among workers in coal-mines—*miner's cachexia*. In this country it is rare, though alleged to have been seen in the Southern States. The importation of infected Italian, Hungarian, and Polish laborers may, at some future time, cause the propagation of the ankylostoma parasite in the United States. The anemia of ankylostomiasis is progressive, and it is noteworthy that no organic cause for it can be discovered. There may be in addition, slight gastro-intestinal disorder (anorexia, colicky pains, nausea and vomiting, and constipation alternating with diarrhea). In cases marked by an acute development of anemia considerable general weakness,

dyspnea and sometimes dropsy may ensue. The areas of the apical cardiac impulse and of cardiac dullness are increased downward and laterally. Various murmurs—hemic—may be heard, and the pulmonic sound may be accentuated (*vide* Pernicious Anemia, p. 429).

**Diagnosis.**—This is made by finding the eggs or mature worms in the feces. The former are oval-shaped, about 0.05 mm. ( $\frac{1}{200}$  inch) in length, and have a much thinner shell than the ova of the round-worm. They do not segment except within the intestine. In any case of pronounced anemia in which the cause is obscure the patient's dejections should be carefully examined for the ankylostoma parasite or its eggs.

**Duration.**—The disease may last for months or for several years.

**Prognosis.**—If left untreated, the affection may end fatally. Intense anemia, obstinate diarrhea, and profound nutritive disturbances constitute symptoms of grave import. Properly treated, the prognosis is quite favorable. A spontaneous cure may occur in some cases.

**Treatment.**—*Prophylactic.*—Workmen in mines, tunnels, and brick-yards, and in tropical localities especially, should be warned not to drink the water close at hand without previous boiling and then cooling.

*Medicinal.*—Anthelmintics to kill the ankylostoma and purgatives to remove it from the intestine are indicated as for other intestinal parasites. The oleoresin of male fern in  $\frac{1}{2}$ - to 1-dram (2.0–4.0) doses, san-tonin, and thymol are very useful for the first-named object.

Cathartics or enemata are used to bring away the dead parasites, after which nourishing food, iron, and tonics are to be given.

## TRICHINIASIS.

(*Trichinosis.*)

The parasite that gives rise to this affection is the *trichina spiralis*.

**Natural History.**—The mature male worm is 0.8 to 1.5 mm. ( $\frac{1}{20}$  in.) long and the female 2 to 4 mm. ( $\frac{1}{12}$ – $\frac{1}{6}$  in.). The head is pointed and unarmed, and the neck is long and more slender than the body, which has a round blunt end. The worm is viviparous. It inhabits the intestines of such animals as the rat, dog, cat, hog, and man.

The embryo or muscle trichina is about 0.6 to 1 mm. ( $\frac{1}{25}$  in.) long, and lies coiled up in a spiral form within an ovoid capsule in the sarcolemma-sheath of muscle-fiber. The life-history begins with the larval state of the trichinae encysted in the muscles. When this flesh is eaten by another animal, or by man, the larvæ are liberated during the digestive process. Passing into the intestines, they reach the adult stage in from two to four days, being then sexually mature, and in five to seven days more they produce hundreds of living embryos.

The intestinal trichinae become fully grown, and then usually die in from four to five weeks. The female trichina may bring forth several broods of embryos during her life-period in the intestine. The living embryos leave the intestine at once, and invade the muscles through various channels—principally along the connective-tissue routes—so that the symptoms of muscular irritation develop in from seven to ten days after eating the trichinous meat. The embryos attain to maturity (larval form) in about two weeks after entering the muscular tissues. Their presence causes a mechanical irritation that results in the formation of a

fibrous capsule in from four to six weeks. In man it probably becomes



FIG. 81.—*Trichina spiralis* from the head of the right gastrocnemius muscle three weeks after the first symptoms appeared (Queen obj.  $\frac{1}{2}$ ; eye piece No. 11).

encysted at a later period than in the lower animals, as shown by the accompanying illustration, taken from a case under the immediate observation of Dr. L. Napoleon Boston (Fig. 81). Usually but a single worm is found within one capsule, though occasionally three or four are seen. Leuckart found numbers of embryos free in the abdominal cavity of infected animals; they have also been found in the mesentery. The encapsulated trichinæ may live many years in the muscles.

With increasing age the capsules

become thicker and may be the seat finally of calcareous infiltration. **Pathology.**—The diaphragm is most thickly infested with the larval trichinæ. Next in order are such trunk-muscles as the intercostals and abdominals, then the muscles of the neck, including the larynx, head, eyes, and extremities. Up to the seventh week of the disease the intestinal trichinæ may be very numerous, as many as a dozen being found in a drop of intestinal mucus. There may be some intestinal inflammation (catarrh) and the mesenteric glands may also be swollen and appear like those of typhoid fever. In cases that proved fatal during the second month, Cohnheim noted an abundance of fat in the liver, a granular state of the renal epithelium and of the heart-muscle, broncho-pneumonic areas (occasionally), and hypostatic pneumonia (frequently). Microscopically, the muscles show "the changes characteristic of acute myositis" (Fitz) after the fifth week. The trichinous cysts in the muscles may be seen with the naked eye as small, grayish-white, opaque, "oat-shaped" specks, longitudinally disposed in the meat-fibers.

**Sources of the Trichina.**—The trichina was first found in pork—the usual source of trichiniasis in man—by the late Joseph Leidy. It should be noted that some individuals may be dangerously infested with trichinæ and yet give no symptomatic evidence of the presence of the parasite. Recent investigations show that the live trichinæ may be found in the fatty as well as the fleshy portion of pork. The pig is infested by eating trichinous rats, trichinous pork, or possibly human or porcine excrement containing the embryos of propagating intestinal trichinæ. The rat may be the original host of the parasites, or it may itself become infected by older rodents eating their fellows, or by eating trichinous pork or human or porcine excrement voided during the stage of intestinal infection.

As to the frequency of the infection of hogs, it may be said that about 2 per cent. were found to be trichinous, according to Salmon's report (1884), of nearly three hundred thousand examinations of American pork. Other examinations, however, show a variation of infection of from .05 to 6 per cent. of hogs. In Prussia, according to Eulenberg's statistics, the ratio is decidedly less varying—from 1 to 2160 hogs (1876) to 1 to



1817 (1889). According to Osler, "the dissecting-room and postmortem statistics show that from one-half to two per cent. of all bodies contain trichinæ."

Of course, man, as a rule, becomes infected by eating raw or partially cooked pork containing living muscle-trichinæ (larvæ). The habit of indulging in raw ham and sausages, so common among the Germans of Prussia (particularly during pic-nics) and in some parts of the United States where German immigrants have settled in large numbers, explains the comparative frequency of this parasitic disease in such localities. Trichiniasis has occurred in epidemic form in North Germany, France, Spain, Russia, the Scandinavian countries, and in several of the north-western United States.

**Symptoms.**—The fact that the *postmortem* examination often reveals the presence of muscle-trichinæ, whereas no history of trichiniasis or of any disease resembling it has been obtainable, shows that one may eat trichinous pork containing a small number of larvæ without the development of any symptoms. It is to be recollected that to the migration of the parasites the principal symptoms of trichinosis are due.

In well-marked cases of infection gastro-intestinal disturbances appear on the second or third day after the ingestion of the infected meat. Vomiting, diarrhea, and colicky pains in the abdomen may be present. The diarrhea sometimes takes on the characteristics of a choleraic attack or may be followed by obstinate constipation.

Extreme "muscular weariness" and bodily fatigue often occur for several days before the embryonic parasites can have begun to wander into the muscles. On about the tenth to the fifteenth day, when migration usually commences, chills, followed by a temperature of  $101.5^{\circ}$  to  $104^{\circ}$  ( $38.6^{\circ}$  to  $40^{\circ}$  C.) and marked myositis, come on. The muscles are stiff, tense, painful on pressure and motion, and somewhat swollen. The flexors of the extremities are particularly sore and often firmly contracted, causing the knees and elbows to be acutely bent. Mastication, deglutition, and phonation may be difficult and painful because of the involvement of the muscles of the jaws, pharynx, and larynx. Intense and distressing dyspnea is frequent on account of the involvement of the diaphragm and intestinal muscles. The temperature shows marked remissions in most cases, and may even be subnormal. The fever lasts from three to seven weeks. The pulse varies with the temperature.

*Edema* is characteristic in nearly all of the cases. It appears on about the seventh day after the infection, and begins in the face, usually being noted first in the eyelids, and extending thence to the extremities and trunk during the height of the muscular symptoms. It may last for several days, then disappear for several days or a week, and reappear. Ascites even has been observed. Edema of the larynx and bronchial catarrh, the latter rarely leading to broncho-pneumonia, may also supervene and add to the gravity of the dyspnea. Profuse sweating may last for several weeks. Miliaria, urticaria, acne, furunculosis, herpes, and pruritus may occur as skin-manifestations. Insomnia, headache, a temporary loss of the tendon-reflexes, and dilatation of the pupils (Rupprecht) have been noted among the nervous symptoms. Prolonged cases show a marked degree of emaciation and anemia.

Complications, as a typhoid state, hypostatic pneumonia, and pleurisy

may appear. Albumin, with casts, and occasionally red and white corpuscles are found in the urine.

*Recovery* is effected in mild cases within two weeks; in the severe cases of infection from six weeks to several months may be occupied before convalescence begins.

**Diagnosis.**—The following symptoms are regarded as pathognomonic: sudden swelling of the face, coming on after the patient has suffered for several days from muscular soreness; loss of appetite, fever, and profuse sweats (Böhler); painful, tender, and “rubber-like” hardness of the muscles, with difficulty in movement; semiflexed extremities; gastro-intestinal catarrh, with a red, dry, coated tongue; dyspnea, diarrhea, and edema of the extremities following the subsidence of that first noticed in the face. Friedreich also emphasizes the hoarseness due to invasion of the laryngeal muscles.

*Meat- and sausage-poisoning* may be distinguished from trichiniasis by the more rapid course of the former, and by the dry throat and skin, jaundice, visual disturbances, more marked gastro-enteritis, and the absence of edema and muscular symptoms.

Direct examination of the passages and of the muscles may be resorted to. The discovery of the parasites in the pork a portion of which has been eaten by the sick of course establishes the diagnosis. A low-power microscope should be used to examine the intestinal mucus for the trichinæ. Light purgation should precede this endeavor. Harpooning such muscles as the biceps for the purpose of removing some muscle-fiber, or directly incising a small portion under Schleich's method of infiltration-anesthesia, may permit of a positive diagnosis in some cases.

*Acute rheumatism, cholera, typhoid fever, and acute polymyositis (pseudo-trichiniasis)* may at times resemble trichiniasis. Epidemics of the parasitic disease are more readily diagnosed than an isolated case.

**Prognosis.**—This depends upon the number of parasites ingested with the infected meat or sausage, and upon the number of embryos generated in the intestines by the matured worms. Marked early diarrhea is favorable. The prognosis should be guarded, however, in all cases, as the mortality-rate may range from 5 to 35 per cent. Death, too, often occurs as late as from the fourth to the sixth week.

**Treatment.**—Prophylaxis is of supreme importance, both as to the infection of the hog and the danger of eating infected pork. Care should be exercised in the feeding of swine, and the destruction of rats should be made as complete as possible in and about the styes. Pig-excrement should be removed and burned, and feeding with milk, bran, grain, and vegetables should be forced upon all keepers of swine.

Rigid inspection of the meat-supply, as is done in Germany, should be carried out by sanitary officers employed by the government. It is held to be an impracticable measure where immense quantities of meat are handled daily to examine microscopically simply a minute fragment of pork taken from each hog slaughtered. Decidedly the safest and most efficient way to prevent trichinosis is to thoroughly salt, smoke, and cook the pork that is to be used. Roasting should be particularly well done, in order that the heat may effectively reach the central portions of the meat. Putrefaction does not kill the parasites.

The treatment of those who have eaten trichinous meat should be by

a prompt evacuation of the bowel, especially within the first twenty-four hours, as after the embryo young have been brought forth and have passed into the muscles no known treatment is successful in attacking them. Calomel is one of the best drugs, and active purgation usually follows its use in large doses, succeeded by salines; rhubarb, senna, sulphur, aloin, and large doses of oil or glycerin may also be tried. In combination with the purgatives some anthelmintic (male fern, santonin, thymol) should be used. The encysted or larval parasites are not accessible to treatment, although picric acid has been recommended. The symptoms to be met are the great muscular pains, insomnia, and weakness, which is often severe in protracted cases. Prolonged hot baths, anodyne embrocations, with hypodermics occasionally, may prove useful for the first; bromids, chloralamid, and the like for the second symptom; and a concentrated liquid diet, strychnin, peptonoids, and the like for the last. Massage, electricity, and stimulating applications, as chloroform liniment, may be required during convalescence and for some time thereafter to combat the muscular weakness, soreness, and stiffness.

#### FILARIASIS.

(*Filaria Sanguinis Hominis*.)

There are several varieties of filariæ that may be found in human blood. The two principal ones are the *filaria sanguinis hominis nocturna* and *filaria sanguinis hominis diurna*. The first is a white, opaline, thread-like worm, tapering toward the ends, which latter, however, are blunt. The male is 83 mm. (3.2 inches) long; the female 155 mm. (6.1 inches). The second worm is known only in embryonic form, and is distinguished by granulations in the axis of the body. Manson found them in the blood of Congo negroes, but only during the daytime. On the other hand, the nocturnal filaria is found only at night, or, if the host be either by habit, necessity, or choice, a day-sleeper, during this time, showing, then, that there is some condition of the body during quietude that is conducive to the appearance of the filaria in the blood (blood and chyle flow, Granville). This "filarial periodicity" is a curious and striking characteristic of these parasites.

The embryos are produced by the female in great numbers, and are so small that they readily pass through the capillaries. According to Manson, who, in 1877, found the larvæ of *filaria sanguinis hominis* in the stomach of a female mosquito, it is probable that after filling itself with the blood of an infested man during sleep, the mosquito seeks stagnant water, dies, and the larvæ are set free. In this way it may happen that man takes in the embryos through the drinking-water. They find a permanent seat in the lymphatics of the human host, mature, and bring forth young, which may again infest the blood by passing through the lymph-ducts into the thoracic duct and general circulation.

The geographic distribution of the filaria is limited mainly to the tropics and sub-tropics. Filariasis is most common in Brazil, the West Indies, Mexico, the Southern States, Southern China, India, Egypt, a part of Australia, and the southern Pacific islands, where it is quite endemic.

The **symptoms** of filariasis are in abeyance until some obstruction



of the lymph-channels is caused by the parasite. There are several conditions or endemic diseases produced. **Elephantiasis arabum** is believed by Manson to be the effect of these parasites in a certain proportion of cases at least. In specimens of night-blood from 88 Cochin Chinese he



FIG. 82.—The movement of a single filaria during a series of four successive instantaneous exposures. The length of each exposure was one-fifth of a second, the entire series occupying less than five seconds. The magnification is to eight hundred diameters, with a Zeiss one-twelfth homogeneous immersion lens (F. P. Henry).



FIG. 83.—Filaria alive in the blood. Instantaneous photomicrograph. Four hundred diameters magnification. Four millimeters Zeiss apochromatic (F. P. Henry).

found filariæ in 21; 14 specimens came from patients with elephantiasis, and only 1 showed filariæ. This latter fact, he explains, is to be expected, since, in order to give rise to elephantiasis (due to an infarction of the lymphatic glands connected with the diseased areas), the adult filariæ must lie on the distal side of the glands, which makes it impossible for the young filariæ to pass into the general circulation. "Therefore the person least likely, in a filarial district, to have filariæ in his blood is one who is the subject of elephantiasis."<sup>1</sup>

**Hematochyluria and Chyluria.**—The patient passes a white, opaque, milky urine, occasionally bloody, with a clotty sediment. This may be intermittent, and normal urine may be passed for many weeks before chyluria or hematochyluria reappears. There may be at the same time a slight degree of polyuria. Under the microscope, fat granules and white and red corpuscles are seen. The lively, wriggling embryo filariæ may also be discovered in the urine, as well as in the blood at night. There is a dilatation of the lymph-vessels in the kidneys alongside of the tubules, and in the abdominal lymph-plexuses. Sometimes a little vesical irritation and straining during urination may be caused by the endeavor to pass chylous blood-clots. The thoracic duct above the diaphragm has been found impervious (Stephen Mackenzie).

<sup>1</sup> *British Med. Jour.*, June 2, 1894.

**Lymph-scrotum and lymph-vulva** have been caused by the filariæ. The parts are greatly swollen, thickened, and contain distended lymphatics filled with a turbid and either milk-white, salmon-colored, or blood-red coagulable liquid that is discharged upon puncturing the varices. The filaria is not always found in the exuded lymph. The inguinal and femoral regions are often enlarged and doughy. An erysipelatous inflammation of the parts is not infrequent in these cases, and may be ushered in by a chill and high fever, lasting a day or two, and ending with a profuse sweat.

The filariæ have also been found in a case of ascites (Winckel), in one of hemoptysis (Yamane, Japan), and, by the same observer, they were found in the feces (chylous diarrhea).

**Treatment.**—Prophylaxis in regard to the drinking-water is essential in order to avoid filariasis. Filtering, boiling, and storing the water in mosquito-proof receptacles is sufficient. Thymol in from 1- to 5-grain (0.0648–0.324) doses, given for from two to eight weeks, has caused the disappearance of the larval filariæ in several cases. Methylene-blue appears also to have produced a cure in a case of chyluria reported by Flint, although Laveran and Henry believe that it is of little value. The latter states that he has “given this drug in larger doses than were used in the case reported by Flint, and for a much longer period, without the slightest effect upon the parasite.”<sup>1</sup> The adult filaria seems to be beyond the reach of any known medication that will not prove dangerous, either directly or indirectly, to its human host.

#### DRACONTIASIS.

(Guinea-worm Disease.)

The parasite is the *filaria* or *dracunculus medinensis* or *persarum*, common in the tropics of Asia, Africa, and America. It is only recently that the male guinea-worm has been found. It is usually solitary, and measures from 50 to 100 cm. (20 to 40 in.) in length and about 2 mm. ( $\frac{1}{12}$  in.) in diameter. It is cylindric, whitish, with blunt papillated head, and a sharp, curved tail. The body is nearly filled by the uterus, which contains innumerable embryos. The live young dracunculus escapes from the intestines of an infested man, ox, horse, dog, or jackal, enters the body of a cyclops or small cray-fish, and there becomes a fully-developed larva. It is then taken into the stomach and intestines of man through the contaminated drinking-water. The female enters the intestines by way of the mesentery, and the male worm, after fulfilling its sexual functions there, probably dies, while the female brings forth its young, which pass into the connective tissues of its human host. The worm has an inexplicable affinity for the subcutaneous and intermuscular tissues of the feet and legs, where it attains full development.

**Symptoms.**—Wherever the parasite is situated, it may often be felt coiled up under the skin, which at that point becomes red, sore, and fluctuating like an abscess. When opened, either surgically or naturally by the worm, the head appears through the aperture. The favorite spot for perforation is the dorsum of the foot, though sometimes it extrudes from

<sup>1</sup> *Med. News*, May 2, 1896.

the legs, occasionally from the thighs, and very rarely from the thorax and abdomen.

**Treatment.**—Prophylaxis in regard to the drinking-water and as to bathing where the intermediary host of the dracunculus—the cyclops—has its habitat is essential for safety.

The active treatment embraces the surgical measures necessary to remove the worm entire and to promote the healing of the irritated tissues. The burrow should be opened, and the worm gradually coiled around a quill or a smooth, cylindric piece of wood until it can be withdrawn without being torn and allowing any embryos to escape into the tissues. Roth claims that after incision the application of compresses of carbolic acid (1 to 15) over the wound causes the worm to be removed in two or three days. Native Indian physicians commend highly the local application of the leaves of the “amarpattee” plant. Asafetida and sulphur have been recommended internally, but without any definite result.

#### OTHER FILARIEÆ.

Among other filariæ that have been found in man are the following: The *filaria immitis*, which causes hematuria and has been found in the portal vein, whilst the ova were discovered in the ureteral and vesical walls; *filaria labialis*, found in a lip pustule; *filaria lentis*, found in a cataract; *filaria trachealis* and *bronchialis*, seen in the trachea, bronchioles, and lungs; *filaria hominis oris*, observed by Leidy in the mouth of a child; *filaria loa*, noticed in the tropics among negroes, its habitat being beneath the conjunctiva.

#### OTHER AND UNCOMMON NEMATODES.

**Eustrongylus Gigas.**—This parasite is exceedingly rare in man, but has been found in many of the carnivora and in some herbivora. It is supposed that fish act as the intermediate host for the larvæ. The worm is enormous in size, the female being from 25 to 100 cm. (10 to 40 in.) in length and from 5 to 12 mm. ( $\frac{1}{5}$  to  $\frac{1}{2}$  in.) long. It is a red, cylindric parasite with blunt-pointed ends. Its most common seat is the kidney, which it may destroy, causing hematuria and, perhaps, the presence of the eustrongylus ova.

**Strongylus paradoxus** has been found in the respiratory organs of the pig and in the dejecta of a pork-dealer.

**Anguillula stercoralis** or **intestinalis** occurs in the stools of certain tropical endemic diarrheas. The parasites are oviparous, and the eggs may be taken through the drinking-water. They have been found in the biliary and pancreatic ducts, as well as in various parts of the intestines. Boiling the water as a prophylactic measure and the administration of thymol or male-fern are to be recommended.

**Echinorhyncus gigas** belongs to the *Acanthocephala* (thorn-headed worms) and infests the intestines of the pig. The larval host is the cockchafer or floral beetle grub. In the only case reported, that of a boy (Lambl), a small echinorhyncus was found in the intestines.

**Echinorhyncus moniliformis** occurs in rats, and one case, that of a Sicilian, has been reported by Calandrucio, in which the ova were found



in the feces. The larval host is probably the *Blaps micronata*. The ethereal extract of male-fern causes the expulsion of the parasite.

## CESTODES.

### ECHINOCOCCUS DISEASE.

(*Hydatid or Bladder-worm Disease.*)

THE *tænia echinococcus* is also called *tænia nana* by Van Beneden, but should not be confounded with the *tenia nana* of v. Siebold, a brief description of which follows this article. It is the smallest tape-worm of our domestic animals, and lives between the villi in the small intestine, especially in the larger breeds of dogs, as the mastiff and Newfoundland. It has a length of from 4 to 9 mm. ( $\frac{1}{5}$  to  $\frac{1}{3}$  in.), and consists of only three or four sections, the last one of which is mature. The rostellum projecting from the small head has thirty or forty hooklets arranged in a double row. Hundreds and sometimes thousands of eggs are contained in the mature segment. The intermediary hosts for the larvæ are rarely man, the horse, and the sheep, and more often the hog and ox.

**Life History.**—The ova, embryos, or the proglottides even, of the adult *tenia* are voided by the dog, and in various ways, to be pointed out later, are ingested by man. The dog first becomes infected by eating the bladders or echinococcus cysts of some animal that harbors the larval form of the *tenia*, and the matured *teniæ* appear in from eight to ten weeks. The liberated six-hooked embryos burrow through the intestinal wall or enter the portal vein; they then pass into the solid viscera, as the liver, into the peritoneal cavity, the muscles, lungs, brain, etc. There they develop into the larval form and cause the formation of hydatid or echinococcus cysts. During the latter process the hooklets disappear.

In the development of echinococcus cysts, about four weeks after the ingestion of the bladder-worm eggs, small nodules appear, about 1 mm. ( $\frac{1}{25}$  in.) in size. In about five months the cyst-walls consist of two layers, an external layer and an inner, granular, parenchymatous layer (or endocyst), containing a clear liquid. As the reaction to the irritation caused by the parasite and its cyst increases, a fibrous investment forms around them. At this time, also, small daughter-cysts, or vesicular buds, form the minor granular layer of the mother-cyst, and contain the heads of the larvæ. They are soon set free, and may themselves give rise to other or granddaughter-cysts in a similar way. These really become the breeding capsules of little cellular outgrowths that form the scolices or heads of future *teniæ*. They show the four sucking disks and a circle of hooklets. Each scolex, when taken into the intestine of the dog, develops into an adult bladder-worm or *tænia echinococcus*. This endogenous mode of cystic growth is common in man (*E. hydatidosus*); but in some of the lower animals, and rarely in man, the daughter- and granddaughter-cysts may develop between the two layers of the primary or mother-cyst, and then extrude (*exogenous* variety; *E. granulosus*). A third variety is the multilocular echinococcus (*E. alveolaris*, Buhl),

affecting principally the liver. A large, hard tumor is seen that on section shows a firm connective-tissue framework surrounding alveoli that average a small pea in size. These alveoli contain small echinococcus cysts with thick, laminated walls. They may contain scolices or hooklets, and sometimes they are quite sterile. The echinococci may be situated in the lymph-channels and bile-ducts (Zenker).

The pure hydatid fluid is colorless, odorless, limpid, neutral in reaction, and has a specific gravity of 1005 to 1012. About 96 to 98 per cent. is water, and sodium chlorid, carbonate, and sulphate, traces of sugar (dextrose), and uric acid are found among the constituents.

Among the changes that an echinococcus cyst may undergo the commonest is that of the *death of the echinococci*, as from diminished nourishment due to intense proliferation of daughter- and granddaughter-cysts. The contents become thickened, putty-like, or granular, and even calcified. Remnants of these obsolete cysts, such as the chitinous substance of the old and outer wall-layer and hooklets, may be found. Traumatism or chemical irritation may also cause the death of the echinococcus and obliteration of the cysts. Sometimes *rupture* of the cyst occurs, with serious consequences to the patient; on the peritoneum daughter-cysts or free scolices may be disseminated and grow. Or *perforation* into the respiratory, digestive, or urinary tracts and discharge of daughter-cysts and hydatid fluid may take place. Lastly, *suppuration* and the formation of large hepatic abscesses may ensue, either spontaneously or on account of septic instruments used for tapping the cysts.

**Etiology.**—Carelessness in the feeding and the keeping of dogs is the primary source of hydatid disease, and the preparing of food where dogs are allowed to roam about, to be petted, and so on, accounts for the majority of cases. Females are more often affected than males, and children and young adults seem to be oftener affected than those older in years.

As regards the *geographic distribution*, echinococcus disease prevails most extensively in Iceland, where man and dog live closely together. In Australia, also, many persons are affected. It is not so common in Europe, Asia, or Africa, and in America it is rare.

**Organs Affected.**—The tenia echinococcus has an undoubted predilection for the liver. Next in order of frequency are the lungs, intestines, perhaps the urinary organs, brain, and spinal cord. The spleen, bones, muscles, the heart, and blood-vessels are involved with uncertain frequency.

**Symptoms.**—**Hydatids of the Liver.**—Unless the cystic tumors compress the portal area or the biliary passages, or invade the neighboring viscera, subjective symptoms may be entirely wanting. Not infrequently echinococcus sacs, partly calcified, have been found *postmortem*, not having produced any symptoms during life. Gradual but progressive loss of flesh and strength with the presence of a fluctuating tumor may be the only symptoms present until late in the disease. If the cysts attain a large size, a sensation of dragging, and of pain even, is often present; as a rule, however, pain is absent throughout the course of the disease. If the tumor displaces the diaphragm upward and compresses the lung, cough and dyspnea result. In some cases the sac has ruptured

into the bronchi, and given rise to cough and to expectoration of the fluid and vesicles.

If the portal veins and bile-duct are compressed, splenic enlargement from passive congestion, ascites, and jaundice will occur, these symptoms being more common when the cysts are multilocular. Rupture may occur into the intestines (colon), into the pleura or pericardium, causing pyothorax or pyo-pericardium, or into the inferior vena cava, causing fatal pulmonary embolism.

Fever is usually absent throughout, unless the contents of the sac become converted into an abscess; then rigors or chills, fever (hectic in type), and sweatings occur, with jaundice (more or less intense) and rapid emaciation.

Not infrequently the cyst-wall becomes partly calcified and the contents are reabsorbed, with an entire absence of symptoms, the patient dying in after years of some intercurrent disease.

When rupture occurs, unless the contents be evacuated through the respiratory or alimentary tract or externally, symptoms of collapse develop and are followed by death.

The **physical signs** give on *inspection* fulness or bulging in the right hypochondriac region, especially if the cyst be single, of large size, and situated anteriorly.

*Palpation* confirms inspection and shows a fluctuating mass or masses. A trembling impulse is felt sometimes on deep palpation, aided by light percussion over the opposite side of the cyst, constituting the so-called "hydatid thrill." This sign cannot always be elicited, but when present is pathognomonic of the disease. The remainder of the liver shows uniform enlargement. The spleen is often palpably increased in size from passive congestion.

*Percussion* reveals, in addition to the hydatid fremitus, an increased area of dulness to the left or posteriorly, depending on the location and extent of the growths. If the left lobe be involved, the line of flatness may extend across the sternum to the left hypochondriac region. If the cysts are multiple and on the antero-inferior surface, the stomach may be displaced toward the left and dulness may extend across the epigastrium; if posteriorly, the pleural cavity may be encroached upon, causing an increased area of flatness upward in the postero-axillary line. Frerichs claims the line of dulness posteriorly in hydatid disease to be a curved one, whose convexity is upward.

*Auscultation* gives, according to Santoni and others, a short sharp booming sound when the tumor is percussed, that may be likened to one produced by striking a membrane stretched over a metallic frame.

**Diagnosis.**—In the entire absence of subjective symptoms and of characteristic physical signs, the diagnosis is impossible. If, however, the cyst be of sufficient size to give fluctuation and the liver be irregularly enlarged, with an absence of fever, pain, and marked emaciation, the disease may be strongly suspected. The only certain demonstration of the condition is the discovery of the characteristic hooklets in the aspirated or discharging contents of the cyst. Among the conditions that may be misdiagnosed for hydatid disease are—(a) Dilatation of the gall-bladder, (b) hydronephrosis, (c) right-sided pleurisy with effusion, (d) syphilis of the liver, (e) carcinoma, (f) abscess, and (g) cirrhosis.



## HYDATID CYST.

Previous history negative, except the companionship of dogs.  
Pain and jaundice usually absent.

Enlargement in any direction, depending upon the location of the cysts.  
Hydatid thrill may be present.  
Less so.

## HYDATID CYST.

The history is negative (*vide supra*).

Urinalysis is negative.

The tumor is most prominent over the hepatic area, and is associated with enlargement of the liver.

The duration is indefinite and uremia rare.

## HYDATID CYST.

The onset is slow; pain and fever are absent.

The presence of a fluctuating mass in the hepatic area, *not changing with the position of the patient*. Hydatid fremitus is present, but no bulging of the intercostal spaces.

Aspiration reveals a clear yellow liquid of low specific gravity, containing no albumin, but chlorids and hooklets.

The disease invariably runs a chronic course.

## DILATATION OF THE GALL-BLADDER.

A previous history of having passed biliary calculi is often present.

Attacks of biliary colic followed by jaundice either are present or enter into the previous history.

Enlargement is always in one direction—downward and posteriorly.

“Hydatid fremitus” never present.

The tumor is somewhat movable.

## HYDRONEPHROSIS.

There is a history of renal calculi or of vesical inflammation.

Urinalysis reveals evidences of renal disease.

The tumor is most prominent in the flank and iliac fossa. If extending to the right hypochondriac region, it *does not* move with the liver.

The duration is short; a termination in uremia is common.

## PLEURISY WITH EFFUSION.

The onset is sudden, and violent pain is present, with fever and dyspnea.

The presence of effusion, beginning at the base of the chest and gradually extending upward—*changing with the position of the patient* and accompanied by bulging of the intercostal spaces.

Aspiration gives a cloudy, turbid liquid, containing albumin and flakes of lymph with high specific gravity.

The disease generally runs an acute course.

For a differential diagnosis from (*d*), (*e*), (*f*), and (*g*) I would refer the reader to the discussion of the several diseases (*vide* Diseases of the Liver).

**Echinococcus of the Respiratory Organs.**—The lung has been the seat of the larvæ quite frequently, and instances have been noted especially in North Germany and Australia. ♦ The right lower lobe has been the seat of predilection, though sometimes the pleura is the primary source of trouble. There are pain in the chest, cough, dyspnea, perhaps arching of the overhanging thoracic region, signs of a pleural effusion, a tympanitic note above the prominence, hemoptysis, and the pathognomonic expectoration of hydatid disease. The general condition may not be seriously affected. Perforation into the pleural sac by pulmonary echinococci may be followed by empyema, and, later, by perforation of the chest wall. The heart may be dislocated. Compression of the lung may produce gangrene.

The *diagnosis*, in the absence of the characteristic sputum, is to be made from phthisis and a pleural effusion. Their location at the base of the chest may serve to differentiate hydatid cysts from phthisis, as well as the absence of marked emaciation. The characteristic curved upper

boundary of dulness in pleural effusion and the change of the boundary upon changing the patient's position will serve to distinguish this affection. Puncture of any bulging area will determine the character of the liquid. Pleural echinococci sometimes cause great compression of the lung and a barrelling of the chest on one or both sides. The pain may be quite sharp, and the respiratory murmur either distant or altogether absent.

**Echinococcus of the Mediastinum.**—Hare has collected 6 cases of hydatid disease among 520 cases of mediastinal tumors.

**Echinococcus of the Heart.**—Since most of the cases have shown involvement principally of the right side of the heart, the instances of sudden death that have been reported may be readily understood.

**Echinococcus of the brain and spinal cord** should not be confounded with cystic degeneration of the choroid plexuses. The symptoms of cerebral hydatids are those of tumor, persistent and intense cephalalgia, vomiting, psychical disturbances, convulsions, amblyopia, and "choked disk," and sometimes paralysis. Hydatid disease may develop inside the dura mater, or it may penetrate from without and destroy the vertebræ before they compress the cord to a great degree. The symptoms are those of a compression myelitis.

**Echinococcus of the Spleen.**—About 40 cases of involvement of the spleen have been described. The organ may become greatly enlarged and be mistaken for that due to malaria, leukemia, etc. The hydatid thrill may be detected.

**Echinococcus of the Kidneys.**—More than 100 cases have been observed, mostly in Germany and France. The cyst may be as large as in hydronephrosis. Many of the cysts are of the exogenous form of growth. As a rule, one kidney only is affected, and generally the left one. Abdominal and thoracic compression symptoms may be caused, and bulging is often present in the lumbar region in marked cases. This may be punctured as an aid in the diagnosis. Rupture into the pelvis of the kidney and the discharge of the smaller cysts may give rise to renal colic and to the discharge of the cysts with the urine. More rarely, rupture of a suppurating cyst may take place in the loin.

**Echinococcus of the peritoneum** is rare as a primary condition. Echinococci have also been located in the bladder, prostate, testicle, ovary, uterus, great omentum, mesentery, arteries, lymphatics, thyroid gland, muscles, bones, joints, parotid gland, orbit, and mamma.

A **multilocular echinococcus cyst** may give rise to a very large, fluctuating, bossellated tumor below the liver; this may simulate colloid cancer, either of the liver or the gall-bladder. Icterus, marked and obstinate, with or without ascites, an enlarged spleen, and a long course without decided loss of flesh, are indicative of this form of hydatid. Fatal hemorrhage may supervene.

A peculiar **complication** of echinococcus cysts is the occasional development of urticaria. It has been noted especially shortly after the puncture of a cyst, and this is somewhat diagnostic when it appears.

The **prognosis** is generally grave both as to life and cure, although some cases of hydatid disease of the liver have lasted for more than ten years.

The character of the changes in the cysts and their mode of termination influence the prognosis. Thus, the occurrence of suppuration

is to be dreaded. Spontaneous cures have been noted in a few instances.

**Treatment.**—As in most of the other parasitic diseases, prevention is more or less effectual, and a cure is difficult or impossible. Infection of the dog should be avoided by preventing its gaining access to possible sources of hydatid disease, as the raw flesh of animals, especially in the form of meat-scrap around slaughter-houses. In order that human beings may not be affected, dogs should not be carelessly handled or allowed to be where they may come in contact with food and drink in any way, whether meat or eggs, vegetables, fruits, or cereals. Cleanliness in keeping dogs and in the proper preparation of food are essential in regions where hydatid disease is prevalent.

Medicines cannot reach the parasites in man, situated as they are in larval form encysted in the various tissues and organs of the body. Whenever the cyst becomes large, accessible, and the cause of troublesome symptoms, surgical measures may be resorted to. Among these are, simple tapping, tapping with aspiration, and with the subsequent injection of various substances (as iodine and zinc-chloride electrolysis), and incision with drainage. Excision of the liver cysts has been practised by Raggi, Pozzi, Tansini, and others, but its practical value is still undetermined.

#### TÆNIÆ OR TAPE-WORMS.

**Natural History.**—Tape-worms are found in the intestine of man, and are the matured or completely developed larvæ or cysticerci from the muscles and solid viscera of animals. Different varieties of cysticerci develop from the ova of the respective varieties of tæniæ. These tape-worm eggs, after having passed out of the bowel, may be taken into the systems of various animals by various modes, entering the circulation, it may be, and becoming fixed within the solid tissues, especially the muscles. In about two or three months pea-sized cysts develop, and from the cyst-walls there gradually forms a new tenia-head, called a *scolex*, or nurse. The worm-cysts, popularly termed “measles,” constitute the cysticerci. Remaining in the tissues, they die and become calcified in from three to six years (Strümpell). But, if taken into the stomach by the eating of raw or partially-cooked meat, a tape-worm develops from the scolex. The maturation of the segments of the tape-worm commences several months after the fixation of the scolex in the intestine. In the natural life-cycle of a tape-worm the usual order of lodgement may be reversed. Thus man instead of a lower animal may become the host of the tenia eggs, which in turn may find their way into the solid viscera and muscles to develop into cysticerci. Again, this same order may in some way be brought about by “auto-infection.” The tape-worm, as its name indicates, has a ribbon-like form; although it has a number of segments and joints, giving it a link-belt appearance. When matured, these segments, or *proglottides*, develop male and female generative organs.

**Varieties.**—*Tænia Solium* (*Pork Tape-worm*).—This worm is seen much less frequently here than in Europe. It develops in the small intestine after the ingestion of raw or underdone “measly” pork. This worm does not necessarily exist singly, as its name would indicate,



although such is usually the case. It ranges from 2 to 4 meters (6 to 13 feet) in length. The head is rounded, pin-head in size, and is succeeded by a thread-like neck and by gradually shortening and widening segments. Four suckers and a projecting circle of twenty-six long and short hooklets arm the head of the tenia. There may be as many as 800 segments. The mature ones become detached continuously, and are passed with the feces, several, as a rule, occurring together, and not singly, as in the case of *tænia saginata*. They are about 1 centimeter ( $\frac{2}{5}$  in.) in length and from 6 to 8 millimeters ( $\frac{1}{4}$ – $\frac{1}{3}$  in.) in breadth, and about 1 meter (39.36 in.) from the head they are "approximately quadrilateral" in shape. These proglottides are bisexual. The female matrix occupies the middle of each proglottis, and is provided with from eight to fourteen irregular, tree-like branches on each side. The male generative organs are small vesicles in the anterior portion of the segment. The sexual opening is situated on one side, near the middle. The ovarian or uterine apparatus of a mature segment contains myriads of thick-shelled eggs, each one of which has an embryo with six hooklets.

***Tænia Mediocanellata* (*Saginata*).**—The beef tape-worm is sometimes called the "unarmed tape-worm," since the head possesses sucking disks, but no hooklets. It is more common in this country and even in some of the European nations, as England. Longer than the tenia solium, being 4 to 6 meters (12 to 20 feet) in length, its segments are also thicker and larger, measuring from 16 to 18 mm. ( $\frac{2}{3}$  in.) long, and from 8 to 10 mm. ( $\frac{1}{3}$  in.) broad. The head of the worm as well as the ripe ovum is also slightly larger and proportionately thicker. The ovarian branches are more numerous (eighteen to thirty in number) and divide more dichotomously than those of tenia solium. Proglottides are also found in the stools, where they sometimes exhibit a crawling motion that has caused them to be mistaken for individual parasites. *Cysticercus saginata* has never been observed in man.

***Bothriocephalus latus* (*Fish tape-worm, Tænia lata*)** occurs most commonly in Russia, Switzerland, Holland, and the German Baltic provinces. It is the longest cestode, measuring from 6 to 10 meters (20 to 30 feet). The head is club-shaped, unarmed, and has two lateral longitudinal grooves as suckers. The segments may be distinguished from those of the preceding varieties named by their marked breadth and shortness, also by the centrally situated, tortuous ovarian rosette, and the sexual orifice near the center of the abdominal surface of each proglottis. The ova are larger than those of the pork and beef tape-worms, though thinner-shelled and with a sort of lid at one end. They develop only in fresh water. From them is formed an embryo with vibrating cilia and six hooklets. Pike and other fish swallow these embryos, which develop into cysticerci in the muscles, peritoneum, and solid viscera. The eating of measly fish, raw or partially cooked, thus favors the development of this tape-worm in the human intestine.

**Symptoms.**—Contrary to what has been supposed in days gone by, there are no absolutely diagnostic symptoms of the presence of tape-worm that can be relied upon. Indeed, the existence of a tape-worm in the bowel may not be suspected even because of the total absence of indicative, subjective sensations. On the other hand, teniæ may cause considerable local distress and impairment of the general health. Because of

this fact a knowledge of the existence of tape-worm in certain neurotic subjects leads to an inordinate description of symptoms that exist mainly in the workings of a morbid imagination.

*Alimentary symptoms* of tape-worm may be as follows: anorexia alternating with a voracious appetite, constipation alternating with diarrhea, colicky pains in the abdomen, indigestion, nausea, and vomiting, and sometimes salivation.

*General symptoms* of the teniæ may be added, as lassitude, inappetence, mental uneasiness, worry and irritability, depression of spirits, some physical prostration, and even emaciation. Various *reflex symptoms*, such as pruritus of the nose and anus, vertigo, migrain, tinnitus aurium, palpitation, visual disturbances (even temporary amaurosis), dilatation of the pupils, choreic movements, and epileptiform convulsions have been attributed to these parasites. But, on careful inquiry, adequate causes for some of these symptoms may be found in other associated morbid conditions.

**Diagnosis.**—This is always to be made by the discovery of tenia segments or ova in the underclothing or stools. The doubtful presence of suspected tape-worm may be cleared by the administration of a suitable purgative, which will usually suffice to bring away portions of the worm in the dejections. I would here add a special warning lest mucous casts or shreds or vegetable structures (as of onion) be mistaken for tape-worm.

The diagnosis of the variety of the tape-worm may also be made by a careful scrutiny of the segments. Those of the tenia saginata are larger and fatter than, and their generative apparatus is unlike that, of tenia solium (*vide supra*).

*Hypochondriasis* can be excluded by repeated examinations of the stools, especially after the exhibition of cathartics, and by the uniform failure to detect portions of tape-worm or tenia eggs.

The **prognosis** is favorable. Indeed, teniæ may exist at all ages and for years without any danger to the patient.

**Treatment.**—*Prophylaxis.*—The way to avoid acquiring a tape-worm is to use none but well-cooked meats; this applies to beef and pork in particular. The use of pure drinking-water is of no little importance also. The proglottides of the tenia should always be burned, and not thrown where they may be taken into the bodies of other animals, as the cow or hog, and then be allowed to propagate. Governmental inspection of the meat-supply in abattoirs should be rigidly carried out in all parts of the country.

*Curative.*—Before administering the chosen anthelmintic, the patient needs to undergo a “preparatory treatment.” This has for its object the starvation of the parasite, so as to weaken, if possible, its hold upon the intestinal mucosa. This is specially necessary in the case of tenia solium, in which the cephalic hooklets are obstinately and firmly fixed to the membrane, and since a cure cannot be said to have been effected unless the head be dislodged with the dejecta. For about two days prior to giving the remedy the patient should be restricted in diet to milk, light soups, a little white bread, and the like. Meanwhile, the bowels should be purged gently once or twice, after a simple enema, to clear away accumulated fecal masses that might prevent the easy discharge of the worm.

In the evening preceding the day on which the drug is to be exhibited, a saline cathartic should be given to empty the bowel of fecal matter as completely as possible. The following morning no breakfast should be allowed, and before noon the selected anthelmintic should then be administered. Some authors assert that if the worm does not come away in a few hours, and an intense sense of pressure is felt in the abdomen, a brisk purge is indicated. To make assurance doubly sure, and if the patient be not too weak, it might be well to order a cathartic as routine practice, within a few hours at the latest.

There are several very efficacious anthelmintic drugs to choose from. Prominent among them is male fern. Given to an adult in doses of  $\frac{1}{2}$  to 1 dram (2.0–4.0) of the ethereal extract, and followed in several hours by a calomel and a saline purge, it usually succeeds in bringing away the tenia. Another valuable remedy is pelletierin, the active principle of pomegranate, dose  $\frac{1}{2}$  to 2 grains (0.0324–0.1296) in capsules; or, a decoction of the pomegranate bark may be used, in combination with male fern, as in the Leipsic formula (Strümpell):

R $\bar{y}$ . Granati radicis corticis,	ʒiv–v (128.0–160.0);
Aquæ,	Oij (1 liter).
Mix and macerate for twenty-four hours,	
and boil until reduced to	f ʒv (148.0).
Add: Oleoresinæ aspidii,	ʒj (4.0).
Sig. To be taken in three or four doses, at short intervals.	

Pepo in emulsion or in a sugary paste (about two ounces—64.0—and deprived of the envelopes) is at once a useful and harmless remedy.

Another effective vermifuge is koussou (*Brayera anthelmintica*). An infusion of half an ounce (16.0) of the flowers to one pint of water and mucilage of acacia is made, a wineglassful of which may be taken every half hour. The Germans recommend sometimes the agreeable, though more expensive, Rosenthal's "koussou tablets." Enough of these to make 15 grains (0.972) may be taken within one hour, with *café noir* or lemonade. Koussin (the active principle) in doses of 30 to 40 grains (1.94–2.592) has also been recommended, but should not be given to pregnant women, as abortion may be produced. Among other remedies of value as vermifuges may be mentioned kamala (1 to 3 drams—4.0–12.0—of the powder and hairs, in wine or water), oil of turpentine ( $\frac{1}{2}$  to 2 ounces—16.0–64.0—in emulsion or milk), and thymol. The combined use of such drastics as croton oil renders the action of the anthelmintic drug more certain at times.

Although the head of the tenia may not be detected in the stools along with the body of the worm (and such is usually the case), a cure usually follows nevertheless, since, on account of its smallness, it may easily escape notice, and also from the fact that the head often dies and thus loses its hold upon the membrane, being carried away with the feces. On the other hand, if after the lapse of several months from the removal of a tape-worm, segments again appear in the stools, it may be inferred that the head was not dislodged or that another worm has developed. In cases where the tenia seems to redevelop with remarkable frequency and



obstinacy it may happen that the head and neck are well protected beneath one of the valvulæ conniventes.

After the removal of the tape-worm—a weakening procedure, as a rule—the condition calls for supportive measures. The diet should not be too heavy for a time, but nutritious and easily digestible.

#### TÆNIA NANA.

This is the smallest tape-worm in man (v. Siebold). It varies from 8 to 20 mm. ( $\frac{1}{3}$ – $\frac{4}{5}$  in.) in length and from 0.5 to 0.7 mm. ( $\frac{1}{50}$  in.) in width. The head has four suckers, a rostellum, and hooklets. The segments are yellowish, short, and broad. It is believed by some observers that, occurring in children, as it commonly does, this parasite is the cause of *epileptiform convulsions* and *enuresis nocturna*. Thousands of ova may be found within a cubic centimeter of fecal matter.

#### TÆNIA CUCUMERINA.

(*Elliptica*; *Canina*.)

A small reddish tape-worm found frequently in the intestines of the dog. It is 10 to 40 cm. (4–16 in.) in length. The larvæ or cysticerci develop in the louse or flea of the dog or cat. The parasite is more common in children than in adults, owing to the intimate relation of the former with the last-named pet animals.

#### TÆNIA FLAVOPUNCTATA.

(*Tænia Diminuta*; *Tænia Leptocephala*.)

*Tænia diminuta* is a very small cestode, 20 to 60 mm. ( $\frac{4}{5}$ – $2\frac{1}{2}$  in.) in length, with a small club-shaped head and nearly a thousand segments. The cysticerci inhabit such insects as the *asopia famialis* (caterpillar and cocoon); the *anisolabis annuli* (belonging to the orthoptera); and the coleoptera *axis spinosa* and *scaurus striatus*. Man has been infected a number of times, probably by taking food containing these infested insects.

*Tænia Madagascariensis* and *Tænia serrata* are other forms rarely found in man.

### PARASITIC ARACHNIDA.

**Pentastoma Tenioides.**—This parasite in its adult form is an inhabitant of the nasal fossæ of the dog or horse, though it may also occur in man both in this and in the larval form. The ova are ejected during sneezing, and are then ingested by man. The larvæ are found in the liver, lungs, and kidneys.

**Sarcoptes (*Acarus Scabiei*).**—This insect produces the skin affection known as “the itch,” or *scabies*, an affection more common in Europe than in America, where it constitutes only about 4 or 5 per cent. of all

cases of skin disease. It is most prevalent among the poor and the unclean. The female is visible to the naked eye, and is about 0.5 mm. ( $\frac{1}{50}$  in.) in length; the male is about 0.25 mm. ( $\frac{1}{100}$  in.). Both are nearly as broad as they are long.

The parasite penetrates the skin and lives in a burrow or *cuniculus* that it makes for itself. The female lives in the end of the burrow, which may contain a number of ova, and appears as a minute, brownish-black, dotted, sinuous line, situated chiefly in the cutaneous folds, where the skin is mostly delicate, as between the fingers. Secondary skin lesions, due to scratching, are common. Sulphur ointment, well rubbed in after hot bathing, is usually quite efficacious.

*Sarcoptes scabiei hominis* is a variety of the preceding that infests other animals (cat, dog, cow, horse, wolf, goat, camel, etc.). Occasionally it may gain an entrance into man's skin, but dies simultaneously in the human host, although many invasions may occur.

*Leptus Autumnalis* (*Harvest Bug*).—The most common of several varieties is a mite of a reddish color, having six legs armed with claws and sharp mandibles. It arises among low bushes and thus appears about the ankles and legs. It partially penetrates the skin, boring only far enough with its short, thick head to procure nourishment. Artificial dermatitis may be produced by the irritation of scratching. Mercury, sulphur, and naphthol ointments suffice to destroy the parasite.

*Demodex Folliculorum* (*Comedo Mite*).—This minute parasite may be expressed from swollen sebaceous follicles of the nose, cheek, and other parts of the face. It has a worm-like body with very short legs, and is only about 0.2 to 0.4 mm. ( $\frac{1}{60}$  in.) in length. It is not known to produce acne, as was formerly supposed.

## OTHER PARASITIC INSECTS.

### PEDICULOSIS.

#### (*Phthiriasis*.)

Lice or *pediculi* live on and attack the skin. Three forms are found on man: *pediculus capitis*, *pediculus corporis*, and *pediculus pubis*.

The *pediculus capitis* is whitish or grayish in color, about 1 mm. ( $\frac{1}{25}$  in.) long (male), and has six legs under the front part of the body. The oviparous female is nearly twice as long as the male, and lays from fifty to eighty eggs on the hairs within a week. These ova, or "nits," mature in from three to eight days. Itching is the most prominent symptom, and an eczematous eruption above and behind the ears and in the neck is often associated. "*Plica polonica*" was a phrase once used to designate the matted condition of the hair in extremely dirty, crusty, and long-neglected cases of head-lice.

*Pediculus Vestimentorum* (*Corporis*).—This louse inhabits more often the clothing than the body itself. It is larger than the head louse, and, like the latter, moves slowly. The nits are found with difficulty on the

fibers of the underclothing. It sucks blood through a proboscis inserted into the sweat pores, and after withdrawing leaves a minute hemorrhagic speck. Irritation of the skin is produced, and in old cases, as in filthy tramps (the great unwashed class), the skin becomes scaly and quite pigmented (vagabond's disease). The efforts at scratching are almost frantic, and after a cure is effected parallel white lines, the remains of scratch-marks, followed by atrophic changes, may be visible, as in a case that I reported.<sup>1</sup>

**Pediculus or Phthiriasis Pubis (Crab-louse).**—This parasite is not limited to the pubis, but attacks also the hairy region in the axilla, on the chest, and may even reach the beard and eyebrows. It clings firmly to one or two hairs close to the skin. Its six legs with strong claws are placed closely together at the anterior part of the ovoid body.

**Treatment.**—The hair should be cut short where the head-lice and nits are abundant. Saturating the hair and scalp with kerosene oil for twenty-four hours usually kills the parasites. *Body-lice* may be destroyed by scalding the underclothing and hot-ironing carefully about the seams. A hot soap-and-water bath is sufficient for the body, and sedative and antiseptic ointments may be useful adjuvants. Mercurial and beta-naphthol unguents usually suffice in treating for *pediculus pubis*. Prof. J. V. Shoemaker<sup>2</sup> affirms that naphthol is a remedy that seems to meet the indications presented by the three forms of the disease; he prepares it as follows:

Ry. Beta-naphthol,	5j (4.0);
Cologne water,	f3iv-vi (120.0–178.0).—M.

**Cimex Lectularius or Bed-bug.**—This too well-known parasite is flat, brownish-red in color, and from 2 to 5 mm. ( $\frac{1}{12}$ – $\frac{1}{5}$  in.) in length. It infests beds and public vehicles, emitting a disagreeable odor. It is a blood-sucker, and causes considerable itching, local irritation, and urticaria even in some persons, while others are unmindful of their attacks. Sulphur fumigation and mercuric chlorid applications to the harboring places of the bed-bugs are effectual destructive agents. Saturated sodium bicarbonate solution will relieve the burning and itching.

**Pulex Irritans (Common Flea).**—This "ubiquitous" parasite is from 2 to 4 mm. ( $\frac{1}{12}$ – $\frac{1}{6}$  in.) in length, black or (when filled with blood) brownish-red in color, having six legs, the hind ones of which are relatively very large and powerful, enabling it to jump many times its own height. A flea's bite causes a sharp sting, and leaves a slightly raised red spot. Treatment is the same as for the preceding insect.

**Pulex Penetrans ("Jigger").**—This parasite, also called "sand-flea," is indigenous to the West Indies, South America, and the Southern States. The impregnated female penetrates the skin, and especially that of the feet, for purposes of ovulation. As the distention with the eggs occurs, swelling, pain, and even ulceration may appear. The sand-flea is a small, egg-shaped insect, about half the size of an ordinary flea, brownish in color, and exceedingly resistant to crushing force. *Prophylaxis* in regard to foot-wear is necessary. Essential and antiseptic oils may also be put on the feet or stockings.

<sup>1</sup> *International Clinics*, vol. iii. third series, p. 76.

<sup>2</sup> *A Practical Treatise on Diseases of the Skin*, p. 849.



**Ixodes** (*Wood-tick*).—There are several varieties of tick- or wood-louse that may attack the human skin, among which *ixodes albipictus* is supposed to be the most common. *Ixodes ricinus* and *ixodes bovis* are found on horses and cattle. They are blood-suckers, adhering to the skin very firmly, and wheals may be produced by them. A drop of turpentine, or of some such essential oil as anise or rosemary, will cause them to loosen their hold.

**Dermanyssus Avium et Gallinæ**.—These bird- and fowl-insects are small and grayish-white in color, and may attack the human skin and cause eczematous eruptions, owing to the scratching induced by the irritation.

**Culicidæ** (*Mosquitoes and Gnats*).—The blood-sucking mosquito (*culex auxifer*), so well known, may also transfer to human beings the filaria sanguinis hominis and perhaps the plasmodium malarie.

The gnat (*culex pipiens*) is very troublesome during certain seasons, particularly along water-courses and in wooded districts. Its bite is quick, sharp, and stinging.

The **hirudo** (leech) is a parasite that sometimes attaches itself to bathers. In the tropics it has been known to cause severe bites and inflammation.

The bites and stings of bees, wasps, spiders, and ants have been known to cause considerable inflammation, edema, and blood-poisoning.

**Œstridæ** (*Bot-flies*).—These may become parasitic in man in the larval form. Species of the *hydoxerma* and *dermatobia*, that infest the skin of the horse, ox, goat, etc., have also been observed among the Central and South American Indians. They burrow beneath the skin of the abdomen, scrotum, and other regions.

**Muscidæ** (*Common Flies*).—Common flies affect the skin of man by depositing eggs in wounds. The ova hatch within twenty-four hours sometimes, and the dipterous larvæ may swarm to make the so-called "living" wound or sore (*myiasis vulnerum*). The larvæ or maggots do not penetrate the tissues, however. The principal flies that infest wounds are the flesh-fly (*sarcophila carnaria*), the blow-fly (*calliphora vomitoria*), the screw-worm fly (*compsomyia macellaria*), and the ordinary house-fly (*musca domestica*).

Internal myiasis may also be caused by swallowing the ova of these flies. The larvæ may thus be vomited or defecated.

Epidemic urticaria is often caused by the migration of the caterpillar (*cuthocampa*). Among other parasites that attack man and inhabit particular regions are the following: The *simulium reptans*, or creeping gnat of Sweden; the *seroot-fly* (*zimb*) of Abyssinia; the *ixodes carapato*, a virulent bed-bug in Brazil; the *hæmatopota pluvialis* (Clegg) of the West Highlands.



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